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TRANSACTIONS

OF THE

SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

VOL. XII. -15

1918-19. -12

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SPECIAL NOTICE.

The next Meeting of the Society will be held on Friday, 17th January, 1919, at 5.30 p.m.

Tea will be provided for Fellows between 5 and 5.30 p.m.

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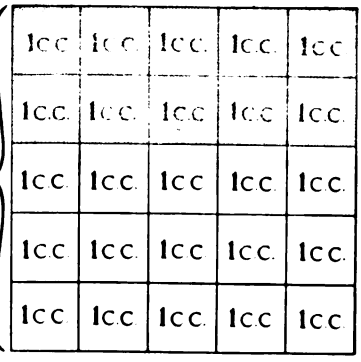

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The Editor of the TRANSACTIONS will be pleased to receive short papers to be included under the heading of "Notes and Comments," or "Questions," in addition to longer papers on subjects of Tropical interest, which may be offered for presentation at meetings of the Society. He will also be glad to receive books for review.

Attention is drawn to the rule that Contributors of papers to the TRANSACTIONS receive 25 reprints free of charge, *provided they apply for these when sending the paper.*

Any communication for the Editor should be addressed to THE EDITOR of the TRANSACTIONS of the Society of Tropical Medicine and Hygiene, 11, Chandos Street, Cavendish Square, W. 1.

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TRANSACTIONS

OF THE

SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

OCTOBER 18TH, 1918.

VOLUME XII. No. 1.

Proceedings of a Meeting of the Society held on Friday, October 18th, 1918, at 5.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Surgeon-General Sir DAVID BRUCE, K.C.B., F.R.S. (*President*), in the Chair.

THE SANITARY ASPECTS OF WARFARE IN SOUTH-EASTERN EUROPE.

BY PROFESSOR W. J. R. SIMPSON, C.M.G., M.D.

THE EXCELLENCE OF THE MEDICAL AND SANITARY ARRANGEMENTS IN FRANCE.

I propose to bring before the Society a general survey of the sanitary aspects of warfare in South-Eastern Europe rather than any details regarding my individual experience in the Balkans.

Until the treaty of Brest-Litovsk there was in this war a Western and an Eastern front in Europe. There is now a Western and a South-Eastern. Throughout the war the Western front has been distinguished for the wonderful success of its medical arrangements. In no former war have such brilliant results been attained in the surgical, medical, nursing; administrative, scientific and sanitary sections.

The splendid organisation of the hospitals, the transport of the wounded and sick, and the remarkable results which have attended the treatment in hospital of the sick and wounded have secured the unqualified admiration of those who have studied them. Similarly the comparative freedom from epidemic disease owing to the sanitary administration has rendered the four years' war unique in the history of wars in France. Side by side with these there have been scientific investigations which have added much to our medical knowledge, among which may be mentioned the infections of wounds and their prevention and treatment, and the research into trench fever.

THE SANITARY PROBLEM MORE COMPLEX IN SOUTH-EASTERN EUROPE.

When we turn to the South-Eastern front we find the same excellence in the British hospital and medical arrangements, although in an area far from England, with its corre-

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sponding difficulties of transport and supplies accentuated by the nature of the countries concerned. The campaign, however, is conducted in areas subject to great ranges of temperature between summer and winter, and where, owing to the domination of the Turk for over 500 years, the inhabitants live under more or less primitive conditions, without any special organisation for or attention to the preservation of health.

Wars in this region have always been distinguished for much sickness and mortality, and it is because I think that information gathered from what has already happened may be useful in securing the adoption of more effective measures in these and in tropical countries, where the sanitary problems are much more complex than in France, that I am reading this paper.

South-Eastern Europe the Gateway of the East and West.

The South-Eastern corner of Europe has always been a great fighting arena. It is the gateway on land to the East and for the West, and as such its possession has been coveted in turn, and fought for by the rulers of expanding empires on either side of it from the earliest times. The last of the great Powers in possession is the Turk. For the past 200 years Austria or Russia, alone or together, have attacked Turkey with the object of taking her place, and they have considerably reduced her hold. Now it is the ambition of Germany, in alliance with Austria, to become heir to the Turk.

Germany remembers that Alexander the Great with a well-trained army, and without railways or other modern means of transport, started from the Vardar Valley, defeated the Persians—the most powerful nation of that time—conquered Palestine and Egypt, destroyed the Persian imperial power at Arbela, and extended his conquests to Afghanistan and India. It was only war-weariness of the soldiers, after nine years of incessant warfare, that prevented him from adding India to the Macedonian empire.

Germany hopes, and I do not for a moment believe she will discard that hope, whether as an empire or a republic, that she will one day be in possession of the gateway, or so control it that she will be able with her Allies to attack the British Empire at a vulnerable spot and threaten our possessions in Africa and Asia. If she cannot retain the gateway in this present war by force, nor by diplomacy cheat the Allies, she will, I think, unless great foresight be shewn, endeavour to do it in times of peace by colonisation and bide her time. It is not without purpose that the Serbian population has been largely exterminated and scattered. It makes it easier to colonise and to occupy by peaceful penetration.

Sickness and Mortality in the Early Wars.

Although the chief interest necessarily centres in the sickness and mortality that have occurred in the war of to-day, yet some of the events in the more remote times are not without their lesson. For instance, it is germane to the subject to recall the fact that the immense army of Xerxes on its retreat through Thessaly and across the Vardar Plain, and that portion of the country now occupied by the British troops in the Struma Valley, suffered from hunger, dysentery and pestilence to such an extent that the greater part of it perished, and when the remnant reached Abydos the major portion died, though food and water were plentiful. In the accounts of the wars between Austria and Turkey and between Russia and Turkey in the eighteenth and early part of the nineteenth century, the invariable story is one of much sickness and mortality from intermittent fevers, dysentery, typhus fever and plague, and from the effects of a bad commissariat. The infection of typhus and plague were not confined to the armies, but spread to the civil population, who, in their turn, infected the soldiers billeted in the villages and towns. It was largely owing to these frequent wars, with their movement of armies and refugees from place to place, that plague continued in South-Eastern Europe for more than 100 years after it had disappeared from Western Europe. It at last disappeared from Turkey in the middle of the nineteenth century. The Crimean war had no plague, but there was the usual prevalence of intermittents, typhus fever, dysentery and scurvy in the armies, and in addition to these cholera. This disease had been brought to Gallipoli with the

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French troops from France, where it was epidemic. It exercised a considerable influence on the course of the campaign, being the cause of the French army having to retreat from the Dobrudja. The disease spread from the armies and caused great havoc among the civil population.

In the Russo-Turkish war of 1878 9 the Russians lost by death from typhoid, typhus and relapsing fever some 24,000 men out of an army of less than 400,000, while dysentery and intermittent fevers prevailed as usual. The Turkish troops suffered to a corresponding degree.

Disease among the Serbs in the Balkan Wars of 1912–15.

The three Balkan wars, beginning in 1912 and not yet ended, have likewise maintained the bad reputation for unhealthiness which has always distinguished wars in this region.

In the first war in 1912 between the Balkan States and Turkey we heard in England much of the doings of the Bulgarians and Greeks against the Turks, but little of the important victories gained by the Serbs, who signally defeated the Turks at Koumanovo, in what is now considered the decisive battle of the campaign. The Serbs sent 30,000 of their men to Adrianople, and by their superior artillery materially assisted the Bulgarians to capture that city. They moreover defeated the Albanians and Turks, followed their retreating armies in the depth of winter through the mountains and inhospitable wilds of Albania, conquered that country, entering Scutari and Durazzo, and came into possession of all that region which formerly belonged to ancient Serbia. In doing so, this portion of the Serbian army came in contact with the typhus fever which is endemic in Albania, and brought the infection back with them and with their prisoners to Serbia, where for many years past it had been unknown.

The first Balkan war, however, was a short one, and there was little time for the appearance of the usual war pestilence to any great extent. There was, however, sufficient time for typhoid fever and dysentery to prevail, and there is also the outstanding fact that the prevalence of cholera among Turkish troops in the Chatalja lines and in Constantinople, together with the check to the Bulgarians inflicted by the Turks, helped in some degree to prevent that city falling into the hands of the Bulgars. Some 18,000 cases of cholera occurred in the Bulgarian troops, but energetic measures were taken by the Bulgars—under foreign direction—to prevent its extension.

The second Balkan war was also short. Bulgaria, not content with the division of the spoils and hankering after Macedonia, suddenly, without warning, attacked the Serbs and Greeks. The war began on the 30th of June and ended in August with the defeat of the Bulgarians. Cholera, however, which had not affected the Serbs in the previous campaign, now attacked them, and in a short time 7,000 of their soldiers succumbed to this disease. I am informed there is an interesting history attached to this outbreak. Before the war a distinguished Austrian professor was consulted as to the value of inoculation against cholera, and his opinion was such that the Serbians did not feel justified in employing this prophylactic. Nevertheless the Austrian army was inoculated against cholera, the professor having changed his opinion later.

It is interesting to note that in 1916 the tetra-vaccine prepared by Castellani, and consisting of typhoid, paratyphoid A, paratyphoid B, and cholera bacilli, was used by the American Red Cross on a large scale in Serbia and was officially adopted by the Serbian army, and is now used to this day with good results.

Before Serbia had time to recuperate after the Turkish and Bulgarian wars she was plunged into a third. The new arrangements in the Balkans interfered with Austrian and German plans of extension and control in the East. On the 28th of July, 1914, Austria declared war against Serbia. Taken more or less by surprise, the Serbian army had to cede territory where invaded in order to prepare to meet so formidable a foe. The invasion was announced to be a punitive expedition, and under this name Austria inflicted cruelties and atrocities on the civil population which remained behind equal to if not worse than those of the Germans on the Belgians. The triumph, however, was short lived. The Austrians were badly defeated and driven out of Serbia with a loss of 20,000 prisoners.

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In a second offensive the Austrians were again defeated and 4,000 prisoners taken. In a third invasion in November, 1914, the Austrian army succeeded in reaching Valyeyo, but shortly afterwards the Serbs won a great victory over the Austrians, drove them out of the country a second time and captured some 70,000 prisoners and an immense quantity of booty. This was in December, 1914. This marvellous success over an army exceeding five army corps in strength was, however, followed by an event disastrous to the Serbs. When the Serbian army and the civilians who had fled from the invaded territory reoccupied the towns and villages which had been in the hands of the Austrians there sprang up an epidemic of typhus fever, which soon spread in every direction and attained an immense magnitude. The previous infection of typhus fever from Albania, which had created no alarm, was now heavily reinforced by a strong infection from the Austrian soldiers, who had brought it with them from their own country. According to Colonel SOUBBOTICH, who read a paper before the Epidemiological Society in January of this year, the whole Serbian country became flooded with typhus, and although the epidemic was heroically dealt with by the British, American, French, and Russian missions sent out to assist, in which Dr. ELSIE INGLIS, Colonel WILLIAM HUNTER, A.M.S., and Dr. RICHARD STRONG, of the American Sanitary Commission, rendered such excellent service, at least 150,000 of the Serbian soldiers and Austrian prisoners were attacked, of whom not fewer than 30,000 died. Out of 350 Serbian doctors 126 succumbed to typhus fever, the death rate being 36 per cent.

Colonel SOUBBOTICH mentions one of the military hospitals, which in peace time had only beds for 400 patients, receiving 1,600, of whom 1,000 were suffering from typhus fever, and in which, out of a staff of 42 doctors, dispensers, auxiliary doctors, dressers, and nurses, only three escaped infection. It is impossible to estimate the number of the civilian population that was affected and died of the disease. He states that the disease was recognised as typhus fever in December, 1914; it continued in full force through January, February, and March of 1915; in April it died down and ceased in June. This epidemic, like all epidemics of typhus, was seasonal in character. The cessation in June is an interesting epidemiological fact. With typhus fever was associated an epidemic of relapsing fever, but these were not the only epidemics that had to be contended with. An epidemic of sand-fly fever is recorded as having occurred in August, followed in September by an epidemic of malaria, which Colonel CASTELLANI describes as having a very protean character, a large number of cases being of the comatose type. He records also an epidemic at Geogheli in September, 1915, which was reported to be typhoid fever, but 90 per cent. of the cases of which, when investigated by Dr. STRONG, of the American Sanitary Commission, Dr. JACKSON and Colonel CASTELLANI, turned out to be subtertian malaria of a typhoid type. This reminds me of an incident that came under my own observation. A company of French soldiers came from the environs of Monastir to Sorovich to rest, and were shortly attacked by what was diagnosed as dengue. The result of a microscopical examination of their blood shewed them all to be suffering from malarial fever of the benign tertian type.

SICKNESS IN BRITISH ARMY IN GALLIPOLI.

While these events were happening in Serbia, disease was seriously affecting the British and French forces in Gallipoli. Of British troops alone, over 96,000 had to be removed from the peninsula from sickness in less than seven months, the great majority of these occurring in August and September.

The actual figures relating to casualties were stated in Parliament on December, 1915, as follows :—

DARDANELLES.

	Officers.	Men.
Killed ...	1609	23670
Wounded	2969	72220
Missing	337	12114
	4915	108004
Sick (all ranks)	...	96663

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This number of sick did not include those who remained on the peninsula in spite of being ill.

It may be of interest here to note that the total number of sick in the Boer War for the 2½ years was 63,644. It has always been held that the sickness in the Boer War was abnormal.

Sickness and Mortality during the Serbian Retreat.

At the time of the sickness at Gallipoli, the Russian armies, needing food and military supplies, had been forced by Mackensen's victories to retreat from the Carpathians. This retreat exposed Serbia later to a fourth invasion of the Austrian army. The Serbs, weakened by the typhus epidemic and the previous campaigns, were in an ill condition to resist with any possibility of success an attack by the Austrians and Germans under the leadership of Mackensen on the one side, and by the Bulgarians, who had now joined the Central Powers, on the other. The Serbs, foreseeing that they would thus be attacked, had asked the Allies for 250,000 men; Venizelos was content if the Allies would send 150,000. In October, 1915, the Allies began to arrive, and mustered at first 22,000, and others quickly followed. They advanced to the rescue of the Serbs, who were putting up a stout resistance against overwhelming odds, but they were too few to pierce the wedge which the enemy had placed between the Allied and Serbian armies, and all prospect of a junction was destroyed. The Serbs had now the choice to surrender or retreat through Albania to join the Entente later. The latter choice was made, and then began that memorable and tragic retreat in which the Serbian armies left their country, marched through a hostile and mountainous country in the depth of winter, fighting against their enemies eager to cut them off or destroy them, until they reached the Adriatic, and were thence transported to Corfu. Before Corfu was reached the result of this retreat was the loss of over 100,000 men from hunger, exhaustion, disease and wounds. Less than 140,000 reached Corfu. Of these, even in this haven of rest, where every effort was made to bring them back to health, 10,000 died during the first two months from disease, typhus fever, relapsing fever and famine diarrhœa being the chief. Colonel Castellani, who was lent as Principal Medical Officer to the Serbian Relief Fund, has published photographs of the condition of some of these soldiers. Here is one. They resemble the state of many of the children of refugees in Macedonia in the Monastir district in 1917, and for whom the Serbian Relief Fund has done so much. After a period of recuperation in Corfu, a considerable Serbian army was able to join the Entente armies in Salonika.

Difficulties Connected with the Salonika Campaign.

A glance at the map will show some of the difficulties of campaigning in the Balkans. It is a country of mountain ranges, deep valleys and fertile plains studded with lakes and marshes, and with rivers and streams that overflow their banks. The plains are separated by precipitous hills and mountains from which spurs stretch out in different directions forming subsidiary valleys; the passes between the plains are often narrow and at a considerable height; on the slopes of the hills and mountains watercourses abound, which bring down immense quantities of storm water from the higher ground, and which, meeting, form the tributaries of rivers or flow into the many lakes and marshes. The Struma river flows into the Tahinos lake before it reaches the sea. Besides the rivers, marshes and lakes in the plains there are not infrequently springs coming to the surface, and they in their turn produce marshy ground; near the sea coast, in those parts where the hills or mountains are not far from the sea, springs, lakes and marshes abound. Macedonia is one of the most malarious parts of Europe, the physical features lending themselves to the prevalence of malaria once the infection has been introduced and established. The country has few roads except those that have been made by the Allied forces. From Salonika there are three railways, one passes westward, crosses the great Vardar plain, which is little higher than sea level, and terminates at Monastir, which is over 2,000 feet above the sea level. Owing to the hills dominating this town being in the hands of the Bulgars, the terminus until lately was at Florina, the headquarters of the French command. A second railway goes north and follows the Vardar Valley, and the third also goes north until it

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reaches the Doiran Lake, then eastward to the Rupel Pass—which reminds one of the treachery of the Greek King—there it crosses the Struma river and turns south proceeding to Seres and thence to Drama—all well-known names in the fighting that has taken place in the British sector.

The road to Monastir by the Via Egnatia is more direct than the railway. It passes the Wells of Pella, the only remains except a small part of a thick and massive wall of the capital of Alexander the Great. The water is brought from the neighbouring mountains by aqueducts and conserved in large reservoirs. It is a dreary journey by the road, especially in winter, if the chilling Vardar wind is blowing, until one reaches Vertekop, which is sheltered and where two British hospitals are located. Both have been bombed by German aeroplanes, causing fatalities. In one of these, under the admirable administration of Colonel FRASER CLARK, I had the pleasure of seeing the excellent pathological and diagnostic work that Captain GRAHAM FORBES and his colleagues were doing, and of inspecting the drainage work near to the hospital which he was instrumental in carrying out, and which, joined on to the larger works in the marshes in the plain completed by the French Anti-Malarial Commission, had improved to a very great extent the health conditions of the hospitals.

From Vertekop there is at first a gradual and then rapid ascent to Vodena, a beautifully situated town in the hills, looking down on and commanding the Vardar plain. Water is so abundant that Vodena signifies a place of water. It was another of Alexander's residential towns. Higher up, the road passes by Ostrovo, where the Scottish Women's Hospital, overlooking the Ostrovo Lake, some 1,700 feet high, is situated, then past Kaimachalan on the right, a mountain nearly 10,000 feet high, which was stormed by the Serbs, and which, together with the taking of Gornechevo and Banitza and the range above it, forced the Bulgars to retire from Sorovich and Eksissu. These two towns in 1917 became centres for Russian, British, Serbian and French hospitals. With the capture of Monastir, the front very soon extended from Lake Ochrida and Prespa on the west front, along the range of mountains to Lake Doiran and to the Struma Valley, where the River Struma discharges into the sea on the east.

I spent some eight months in the Monastir district as Principal Medical Officer for the Serbian Relief Fund, whose activities embraced not only medical aid in hospitals and dispensaries established and maintained by the fund for Serbian soldiers and refugees who had fled from their homes, but also the provision of comforts in the form of food and clothing. When I left at the beginning of this year there was one small ambulance hospital at Lake Prespa, a larger one at Monastir, another at the Tcherna bend, a dispensary at Kenali, another at Jevonia, and the principal hospital at Kremyan—at one time this was at Sorovich in Greek territory.

I had exceptional opportunities of seeing and studying the conditions existing in nearly all the sectors occupied by the Russian, French, Italian and Serbian troops; also the state of villages and towns in Greek and Serbian Macedonia. I saw the great work the French Anti-malarial Commission was carrying out. In January of this year I was given facilities by my friend, General SIR MAURICE HOLT, Director of Medical Services of the British Salonika force, to visit the Struma Valley and examine the conditions there, and was asked to make any suggestions which might be of assistance in improving these conditions or in lessening the chances of malarial infection, as he was anxious to do all that was possible to protect the troops. In complying with this request, I submitted a short report which, while giving credit for what had already been done, contained certain suggestions which I understand have been or are being carried out.

The chief towns in Macedonia are Salonika, Vodena, Sorovitch, Florina, and Monastir; the first and last possess very fine buildings, but in Salonika fleas, bugs, mosquitoes, and flies abound, as they do in the other towns. Colonel BALFOUR, in a lecture delivered at Salonika, gave an excellent description of these insect pests. Most of the inhabitants live in villages; some of these, such as Batachin, on the lower slopes of Kaimachalan, inhabited by shepherds, who keep their flocks and cattle away from the village, are clean and the houses well built, but in the majority of villages the inhabitants dwell in houses of a primitive construction, badly lighted and ventilated, and in which

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the fowls, pigs, and donkey occupy one end of the living room and the family the other. When the house is two-storied the family live upstairs and the animals below. It is under these conditions that there is every opportunity for the diseases of animals to be conveyed to man, and *vice versa*. There is one village in which we had a dispensary hospital, and which had been named many years ago the "Empire of the Fleas." Many of the patients admitted to hospital, sometimes suffering from malaria, sometimes with some other dangerous illness, were so abundantly covered with flea-rash that unless carefully examined the impression was given that they were typhus fever cases. The water supply in many places, especially in those near the foot of the hills, is good, owing to its origin from springs, but wherever there are wells they are polluted with filth; latrines, kitchen, and well are usually close together. There is much dysentery among the civil population, both bacillary and amœbic, the former being the most prevalent; but the two diseases which bulk the largest are malaria and tuberculosis. Nearly every illness among the civil population was associated with one or both of these diseases. Among the refugees diseases of malnutrition were common. It was in a country of this description that military operations had to be carried on. Roads had to be made and fighting maintained while the Greek king with an army in Thessaly was intriguing and helping the enemy.

Sickness in the Allied Armies in 1915 and 1916,

Under the circumstances great caution had to be exercised. This meant delay, and delay in a malarious and insanitary country, where the troops undergo great fatigue and are unprotected, is dangerous. By the time Monastir was taken by the Serbs and French in November, 1916, and the British were fighting in the Struma Valley, the toll of sickness from malaria, sand-fly fever and dysentery had been enormous. The British troops perhaps suffered in a less degree than the French except in the Struma Valley, where all the regiments were more or less affected, some worse than others. It is not surprising that the name which the Allied Armies received was the "Phantom Armies." Let me give you one example. A regiment entered the Struma Valley 900 strong. In six weeks there were only 200 left not in hospital.

The French Anti-malarial Commission.

It was in 1916 that the Pasteur Institute recommended to the Government of France that two tropical experts should be sent out to study the malarial situation. This was agreed to, and the brothers Sergent from the Pasteur Institute of Algeria were selected for the mission. As a result of their investigations they reported that it was essential that a Commission—not an advisory Commission, but one endowed with executive powers acting under the Director of the Health Service of the Army of the East to secure the necessary support for its work—should be immediately sent to Macedonia to carry out anti-malarial measures. This Commission, of which Dr. Visbecq was chief, arrived in Salonika in March, 1917, and there was formed under his presidency an inter-allied Commission, with its headquarters in Salonika, to work out and put into practice the necessary measures in their respective spheres. As I had every opportunity of seeing the anti-malarial work that was done in the different parts of Macedonia during the year 1917, I have no hesitation in saying that the inter-allied Commission accomplished a great deal in a very short time, and that its services were of the highest value. Dr. Visbecq, before I left Salonika, kindly furnished me with maps and particulars of the operations conducted by the Commission, and it had been my intention to place these, together with an account of the measures taken, before the Society at the last April or May meeting, had the opportunity presented itself. I shall now only refer to a few details, as the Commission's Report is published in the June number of the *Bulletin de la Société de Pathologie Exotique*, and can be read by all those interested.

The need of the work which the Commission undertook and the systematic way in which it was carried out are shewn by the maps taken from their Report. The first map shews the splenic index of the civil population under fifteen years of age, taken by Drs. Sergent, during the winter of 1916-17, in the principal camping grounds from

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Salonika to the Monastir front and up the Vardar Valley. The percentage of enlarged spleens in some of the localities was 73·2, 61·6 and 56·7.

The second map represents the splenic index in the summer of 1917, as found by the French Anti-Malarial Commission in the towns and villages in that portion of the country in which French, Russian, Italian and Serbian troops were passing through or located.

The other maps shew the same in different parts of the country.

At the same time as these investigations were being made, the more practical work of the Commission proceeded, and consisted in distributing among the regiments and hospitals circulars and post-cards illustrative of the dangers to the soldier of the bites of mosquitoes, providing mosquito nets for bivouac tents and hospitals, issuing veils, carrying out large and small drainage works wherever marshes were close to camping grounds, filling up small excavations close to camps, and occasional oiling, but the latter had to be done sparingly owing to scarcity of oil. Quinine rations were also issued. In the Struma Valley similar measures were introduced. In this valley there was an additional precautionary measure taken, and this was that during the summer the greater part of the troops were withdrawn from the valley to the hills that overlooked it, only sufficient troops being maintained in the infected area to guard the outposts.

In regard to veils, I shewed the Society last year one invented by my wife just before I started for Salonika, and which she calls the "Simpsonette." (It is described in Volume X., No. 7, June, 1917, of the TRANSACTIONS of the Society.) She has greatly improved it. Hoods in future will be made non-inflammable with smokers. All the veils carried about in one's pocket or fastened above the helmet are quite useless as a protection against mosquitoes at night; they are useful to keep off flies from the face during the day, but that is all. I now shew the two kinds that have been found most useful. In future wars in a malarious country I believe the soldier will wear during the evening and night a mosquito headgear, just as he now wears in the Tropics during the day a sun helmet.

COMPARISON BETWEEN THE HEALTH CONDITIONS OF TROOPS IN FRANCE AND THOSE IN SOUTH-EASTERN EUROPE.

I think the account I have given shews that South-Eastern Europe is an unhealthy area for warlike operations, and that though disease has fallen very heavily on the armies of those countries who do not pay the same attention to hygiene as we do, yet in this war the Gallipoli and Salonika experiences demonstrate that the sanitary organisation and measures which have been so effective in France have not been sufficient for armies operating in countries where malaria exists, and where sanitation is in a very primitive state. This state of affairs as regards the preventive side of disease in our armies as distinguished from the hospital and curative side, for which there cannot be but the highest praise, is emphasised also by what happened in Mesopotamia, East Africa, and Egypt. It appears to me that our sanitary organisation for campaigns in tropical and sub-tropical countries must be submitted to a thorough examination, and be reorganised on a very different basis from that which now exists. I gave expression to these views as early as June, 1915, but they were unfortunately disregarded. I repeated them in an article which appeared in the *Journal of Tropical Medicine and Hygiene* of December 1st, 1916, on "The Need of an Organised Application of Tropical Hygiene to Prevent Excessive Wastage from Disease in our Armies in the East," and it was followed later by a letter of mine in the *Times* on the same subject.

These views can be held without in any way diminishing the importance and magnitude of the work done by the British Sanitary Service in France. The results are written large in the health conditions of the army there, and no one realises and appreciates that success more than I do. The reason for my views is that the conditions under which the armies are operating are different, so that what has been found adequate for the West is not so for the East, nor for the Tropics. In the West the armies are waging war—

(i.) In a temperate climate.

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(ii.) The operations are near England and the base of supplies, whether it be food, equipment, ammunition, or other requirements for warfare.

(iii.) France, except in regard to typhoid fever and dysentery, is a comparatively healthy country.

(iv.) There was from the commencement a highly trained medical service for war and a small but well-trained sanitary service to deal with the hygiene of camps, the movement of troops and lines of communication, water supplies were protected and treated, arrangements were made to give the soldiers baths and disinfect their clothing and blankets to free them from vermin, etc., while the beginnings of infectious diseases that might appear were promptly checked.

(v.) The troops were inoculated against typhoid fever and vaccinated against small-pox.

(vi) The only trouble was trench fever, the causes of which were not understood, but in consequence of the researches carried out in France by the American Red Cross Commission, of which Major RICHARD STRONG was Chairman, and by the investigations in this country, under the direction of our President, Sir DAVID BRUCE, the methods of dissemination of this disease have now been made clear. It is hoped, therefore, that a systematic and organised effort will be made to apply this knowledge. Without this application the knowledge gained by the investigation is of no practical value.

(vii.) The sanitary organisation for the troops in France is such as to have attained remarkable success, considering the conditions under which it had to work.

In the East, on the other hand, in Macedonia for instance—

(i.) The climate is extremely cold for a short time in the winter and semi-tropical for many months in the year.

(ii.) The operations are a great distance from England and France and the base of supplies.

(iii.) Supplies of all kind are very difficult to obtain owing to submarine attacks.

(iv.) The area in which the troops are engaged is very insanitary and extremely malarious; the water supplies are polluted, and there is an abundance of fleas, bugs, lice, flies and mosquitoes.

(v.) The armies have suffered from much sickness and mortality, causing a serious loss of effective strength, a great strain on hospital staff, and the need of a multiplication of hospitals, with their attendant expenses, which otherwise could have been avoided.

(vi) This condition of affairs demonstrates that the sanitary organisation for the army is not effective where operating in a notoriously insanitary country, while the experience in Egypt, Mesopotamia and Palestine also demonstrates that it is not sufficient to preserve an army in a state of health in a tropical climate.

(vii.) The International Anti-malarial Commission has done and is doing splendid work to mitigate some of these conditions, but as it began work 18 months after the Allied troops landed, it did not prevent disease in the early and important stages of the campaign.

The arrival of an Anti-malarial Commission 18 months after operations begin, may be advocated on the plea that it is never too late to mend. But certainly, from a preventive point of view, the sanitary officers and staff should arrive if not before, then at the latest with the first staff officers.

The dispatch of this Commission, which was an executive Commission, at least is an acknowledgment that a special working staff to combat malaria was required.

SUGGESTIONS FOR REORGANISATION OF THE SANITARY SERVICES OF THE ARMY FOR UNHEALTHY COUNTRIES AND TROPICAL REGIONS.

It is necessary that the whole attitude towards preventive medicine should undergo a change. To wait until disease and epidemics occur is not prevention, however strenuous the efforts afterward to suppress them may be.

It is merely adopting the same system for a town or district, or regiment or division, as is employed when an individual falls ill. The doctor is called in, and in both cases the method is curative. It is certainly not preventive.

There are certain changes and additions which I would advocate to secure more

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efficient prevention of disease in armies that are destined to fight in tropical and sub-tropical regions and in unhealthy and malarious countries. First of all I am not impressed by the position given to the Sanitary Officer, or to the sanitary service. As rank is equivalent to power in the army, I venture to suggest that the Sanitary Officer of the army should be given a higher position, and that under the Director-General it should be a distinct branch endowed with more executive power.

I suggest for consideration, and I do so because we are now dealing with a national army—

1. That the Director-General should be on the Army Council. In this capacity he would be the Minister of Health for the Army. As a member of the Army Council he would be at once in possession of all the plans that are likely to be formulated, and be in a position immediately to arrange his medical and sanitary services accordingly.

2. There should be two branches under him: the Medical with its different sections, and the Sanitary with its sections. The chief of each branch should have the rank of a major-general. They can always be in close touch with each other in matters which are in common.

3. Each of them should have their Advisory Boards, and on them specialists in their respective departments. The duties of the Tropical Sanitary Board would be the co-ordination of sanitary measures, the recommendation of supplies, the recommending of suitable sanitary officers for the East, and the performance of duties in sanitation similar to those of a general staff, in its preparation of plans for prevention and watching the progress of sickness and epidemic diseases with a view to their prevention. The plans should be prepared in times of peace, and, like the plans of the military staff, should be ready at the first indication of war to be put into operation. Tropical sanitation includes something more than sanitation in cool climates. In order, however, not to multiply Boards, the two representatives of sanitation who were on the Army Medical Advisory Board should be transferred to join the Tropical Sanitation Board.

4. There should be Inspector-Generals of Sanitation for each Army Corps as Consultant Sanitary Officers, and advisers to the Generals and the administrative and executive sanitary officers, and to report on the efficiency or otherwise of the measures employed. They should proceed to the seat of war at the same time as the General Staff, and be in close touch with the Headquarters Administrative and Executive Sanitary Officers.

As regards the higher local administrative and executive sanitary officers, they should be quite distinct from the medical branch. True, much has been and can be done by preventive inoculations against bacterial diseases, such as cholera, the enterics, pneumonia, etc., but the insect-borne diseases need measures of a different order, requiring a very careful and systematic organisation, all kinds of equipment, and a labour organisation; so does the water supply, which is not always rendered safe in semi-tropical and tropical regions by chlorinisation. The food and vegetable supply also needs careful attention. The duties of these administrative and executive sanitary officers involve so much detail and attention to sanitary supplies, equipment, sanitary works, to inspections, consultations and provision of labour, staff and material, that they can never be effectually carried out if combined with other duties. Close touch is necessary, not only with the Statistical, Intelligence, and Bacteriological Departments, but also with the Labour and Engineering Departments, and the Sanitary Department should have depots of their own for the ordinary materials they require.

In addition to the ordinary regimental sanitation there needs to be an organisation for the larger works connected with brigades or divisions, for camps and lines of communication, and for this sanitary officers of rank are required. There is no use sending a captain or a major to a general, and if recommendations are sent through a higher medical officer, possibly not interested in sanitation, they may miscarry.

General sanitation of the district should be quite separate from the duties relating to field ambulance hospitals.

I hope this paper—which purposely has not dealt with details—will be sufficient to give rise to a useful and instructive discussion on the subject by those who have personal experience of warfare in the Near East and in the more tropical regions. We possess the knowledge, but I am convinced we have not the organisation to apply that knowledge in a manner that will safeguard our armies in the East from preventable disease.

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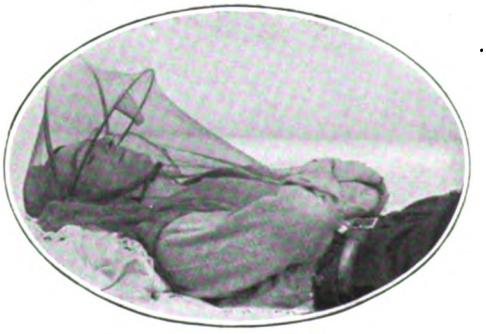
ANTI-MOSQUITO AND FLY PROTECTIVE HEADGEAR (PATENTED).

INVENTED BY MRS. MARY SIMPSON.

No. I.

WORN UNDER THE HELMET.

WORN ABOVE THE HELMET.



WORN LYING DOWN OR ASLEEP.

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ANTI-MOSQUITO AND FLY PROTECTIVE HEADGEAR (PATENTED)

No. 1.

FRONT VEIL THROWN BACK, ACTING AS A PROTECTION FOR THE NECK.

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ANTI-MOSQUITO AND FLY PROTECTIVE HEADGEAR (PATENTED).

INVENTED BY MRS. MARY SIMPSON.

No. II.

WORN UNDER THE HELMET.

WORN ABOVE THE HELMET.

SMOKER TUBE IS USED FOR DRINKING WATER OR TEA THROUGH GLASS TUBE.

WORN LYING DOWN OR ASLEEP.

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FILARIAL INFECTION IN MACEDONIA.

Report of Two Cases of Filaria Conjunctivæ (Addario) in Man, with the First Recorded Discovery of the Male Worm.

BY

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Search into the literature on the subject of Filariasis yields evidence of the extreme rarity of infection by *Filaria conjunctivæ*—a condition confined in its geographical distribution to the south-east of Europe, known to occur in horses and asses, its normal hosts, and incidentally but seldom developing in man.

Records of six examples of human infection exist, and in every case the solitary female worm, usually immature, only has been found.

The first of the two cases to be described provides the only occasion hitherto recorded of the discovery of the male type of *F. conjunctivæ*. Although this parasite, first so named in 1885 by Addario,[1] was obtained from the eye, it is not necessarily restricted to that region; for it has been described in 1906 by Alessandrini[2] as occurring in the forearm, in a case very similar to that recorded here.

The two patients, one of whom—a private in the R.A.M.C., with eighteen months' service in Macedonia—had not previously been out of England, the other a Serbian, came under treatment at the same general hospital attached to the Royal Serbian Army, the former in February, the latter in June, 1918.

Case I.—Pte. J. B., R.A.M.C., one morning, early in December, 1917, noticed irritation, redness and slight swelling on the outer side of the upper part of his left forearm, and, in the skin over it, two small puncture marks, which suggested to him the bite of an insect. During the day this became a definite lump, about the size of a filbert nut. As the lump was quite painless he gave it little further attention at the time, although it shewed no signs of disappearing. Two weeks later the whole of the outer part of the forearm became swollen, hot and red, but still free from pain. It remained so for three days and then subsided, leaving the original nodule as before. This swelling up of the forearm recurred at intervals of about ten to fourteen days, was never painful, and lasted about three days, but did not interfere with his duties as batman.

At the fourth recurrence he applied for treatment. It was decided to wait until the swelling had subsided, and a fortnight later, early in February, he was admitted to hospital under Capt. Lees, M.C., for operation.

On incision through the skin of the upper part of the forearm, a small tumour was found firmly embedded in the subcutaneous tissue overlying the head of the radius and the sheath of the supinator longus muscle, so that it could only be removed by dissecting out from the surrounding tissues. The tumour resembled a lymphatic gland, measuring $1 \times \frac{2}{3} \times \frac{1}{2}$ inch. On incision into the substance of the nodule, a thread-like worm suddenly protruded for a distance of $\frac{3}{4}$ inch. The specimen of both tumour and worm was then sent to the laboratory for detailed investigation.

Two days after the removal of the nodule, the epitrochlear gland from the same arm was excised for microscopical examination. The subsequent course of the case was uneventful. The operation wounds healed by primary intention, and the patient has remained well ever since.

Pathological Report.—Gentle traction on the end of the worm protruding from the

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tumour resulted in its complete removal, but in three portions, the original incision into the excised nodule having divided its length in two places.

*To the naked eye the worm was semitransparent, white in colour, and shewed faint but definite transverse marking. When placed in saline it exhibited slight waving movements for a short time. From the severed ends of the cut portion there poured a copious stream, which created a visible cloudiness of the saline; some of this was collected, and in stained preparations shewed the presence of a number of very small round cells, 3 to 4 microns in diameter, displaying internally a faint cruciform structure, thus ⊂∩⊃∪ in all probability spermatozoa, which in nematodes, Dr. LEIPER informs me, are aflagellate.

After preliminary examination in saline, the worm was dehydrated in solution containing 70 per cent. alcohol and 5 per cent. glycerine, and subsequently mounted in glycerine jelly.

Measurements.—Length, 58 mm. Greatest breadth (towards the head), ·45 mm. (at a distance of 2 mm. from the end). Average breadth, ·35 mm.

Breadth of head, ·2 mm. (at a distance of 1 mm. from the end). Breadth of tail (at the bend), ·25 mm., (at the extreme tip), ·1 mm.

Microscopical Examination: I., of the Male Filaria.—Under the low power by transmitted light the substance of the worm appears of a brownish colour, invested by a transparent cuticle about 8 μ in thickness and composed of an inner and an outer layer. Throughout the greater part of the length of the worm the cuticle shews fine transverse striation, about 1 to 2 μ apart, but not apparent in the portion enveloping the head and the extreme tip of the tail.

Beneath the cuticle, and running in the long axis, can also be seen the longitudinal striation of the dorsal and ventral musculature, by contraction of which the envelope is thrown into shallow transverse folds, giving the faintly corrugated appearance visible, as already mentioned, to the naked eye at the original examination.

The anterior extremity tapers from a width of ·4 to ·2 mm., and is blunt pointed, without obvious papillæ protecting the minute central oral aperture. At this point the cuticle is invaginated to communicate with the fine cuticular lining of the pharynx and its prolongation into the œsophagus.

At a distance of ·4 mm. from the orifice the alimentary tube is deflected laterally in a sharp angular bend to the right, followed by a broad loop to the left, and is then continued on in a fairly straight course, but at various points it is possible to make out the fine coils of either the mid and hind gut or the genital tubules.

On either side of the interior end of the tube are seen small groups of tissue, possibly framework supporting the nerve structures encircling the œsophagus.

The caudal end is curved at a blunt angle towards the cloacal aperture at a distance of under ·1 mm. from the tip, and projecting from the aperture is seen a delicate filamentous spicule, part of the copulatory organ, ·25 mm. in length.

No papillæ whatever are to be made out on the tail.

II., of the Tumour and Gland.—The nodule from which the worm had been removed was examined in the fresh state, but failed to shew the presence of a second or female worm, or evidence of microfilariæ in the wall of the cystic interior.

Sections shew the nodule to be composed of fibrous connective tissue, and round cell infiltration traversed by collapsed spaces, lined with what are possibly the remains of a flattened endothelium.

These spaces may represent original lymph channels blocked by the lodgment of the filaria, and subsequently shut off and enclosed by the resulting zone of inflammatory reaction and fibrous connective tissue formation.

No filaria or microfilariæ could be seen in the sections.

The excised epitrochlear gland revealed nothing abnormal, either in smears from the fresh tissue or in stained sections.

Examinations of the patients' blood were made on several occasions, both by day and

* The drawings submitted by Capt. GRAHAM FORBES have not been reproduced. Specimens were shewn under the microscope at the meeting.

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night, in fresh and stained films, but failed to shew the presence of any microfilariæ, and gave no evidence of leucocytosis.

A differential leucocyte count taken on the day after the operation was as follows:—

Polymorphs ...	...47 0 per cent.	Large lymphocytes...	8·5 per cent.
Large mononuclears...	2·0 „	Eosinophils... ...	3·5 „
Small lymphocytes	38·0 „	Basophils	1·0 „

CASE II.—Pte. Petar Mitrovitch, a Serb, was admitted to hospital on June 27th, 1918, under the care of Capt. McDOUGALL, R.A.M.C., suffering from a swelling of the right side of the nose.

History.—For the past year he had been stationed with Serbian troops in a Turkish village on a part of the Macedonian front. Two months previous to his admission in hospital the right side of the face suddenly became swollen. He described the swelling as white and painless, with a burning and itching sensation over the side of the nose.

The condition of the face subsided somewhat as the result of local applications, but the swelling on the side of the nose persisted.

On Admission.—On the right side of the nose, and just below the right lachrymal sac, was a small, moveable, tense cystic swelling, not involving or adherent to the skin.

There was no evidence of obstruction to the lachrymal duct, and no conjunctivitis or blepharitis.

Operation was performed by Major MILLS, R.A.M.C.T., three days after admission. Incision was made over the swelling, and a small cyst was exposed and dissected out. Its wall was thick and adherent to the underlying tissues; when cut into, a small quantity of opaque glairy fluid exuded from the definite cavity occupying the interior of the cyst, together with a delicate filiform worm, which was extracted, and has been found to measure about $3\frac{3}{4}$ inches (94 mm.).

The skin incision was closed with horsehair sutures and a firm pad and bandage applied. A week later (July 7th) the stitches were removed.

On July 9th a small, hard, moveable swelling, probably an inflamed gland, was noticed on the left side, under the lower jaw.

On July 24th there was a recurrence of the cystic swelling at the side of the operation wound; there was also slight enlargement of the glands on each side just below the symphysis of the lower jaw.

Blood-film examinations, made on two separate occasions, failed to shew the presence of any microfilariæ.

I have at present no further clinical particulars of this case, and it is thanks to the kindness of Capt. LEES, M.C., R.A.M.C., who sent me the notes and the specimens of the cyst and filariæ that I am able to place them on record.

I subsequently sent the notes and specimens of the two cases above described to Dr. R. T. LEIPER, who has very kindly indentified them both as belonging to the same species; in the one case the mature male, the first hitherto recorded, and in the second case, the immature female of *Filaria conjunctivæ* of ADDARIO.

The female specimen is incomplete and unfortunately the head is missing; what remains measures 94 mm., but the character of the finely striated cuticle and the appearance of the blunt pointed tail, leave little doubt as to its sex and species.

The cyst which contained the worm measured $\frac{3}{4}$ by $\frac{1}{2}$ inch, and was too friable to admit of section cutting

Search with a hand lens and films from the inner wall failed to reveal a second filaria or any embryos. Moreover, the absence of embryos from the body of the worm is evidence of its immaturity—a feature also of most of the few recorded specimens of *F. conjunctivæ.*

Measurements—Length (incomplete), 94 mm. Breadth, ·5 mm. Thickness of cuticle, 7–8 μ. Breadth of tail, ·15 mm.

As in the male specimen, the cuticle is transparent and marked by very fine transverse striations, 1–2 μ apart and visible throughout the length to the tail.

Viewed by transmitted light under the microscope the substance of the worm,

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between the cuticular layers on each side is of a brownish colour, and shews the coarse coils of the uterine tubules throughout the greater part of its length crowded with immature ova measuring 2–3 μ.

In addition can be seen the fine longitudinal markings of the muscle fibres.

The tail differs from the male in the absence of spicules, in not shewing any upward curves, and ending abruptly in a blunt point.

The anus is not visible and there are no papillæ to be seen.

The tip of the tail, viewed from the side, shews one of the lateral pits as described in GRASSI'S detailed account[5] of the anatomy of the worm, corresponding to the point at which the cavity of the cœlom pierces the external cuticle and apparently forms a very small short canal measuring 4–5 μ across.

Previously recorded cases in Man.—Though first named by ADDARIO in 1885 as *Filaria conjunctivæ*, this species had been already observed, but not named by DUBINI[3], who found the immature female worm measuring 115 mm. in the eye of a man in Milan.

ADDARIO'S specimen of the female worm enclosed in a nodule, the size of a pea and measuring 95 mm., was extracted by VADELA from the conjunctival sac of the right eye of a woman, aged 70, a native of Catania, in Sicily, in whom a year later a second worm was found in the same eye.

BABES[4] also had described the same species at an earlier date in 1879 and 1880, as occurring encysted in a calcified nodule in the gastro-splenic omentum of a woman, in Buda-Pesth. He named it *F. peritonei hominis*. This specimen was an immature female, measuring 190 mm.

In 1887 GRASSI published a full description of the anatomy of the mature female worm, and named the species *Filaria inermis*, on account of its "unarmed" appearance, due to the absence of papillae from the head.

The female specimens so far recorded in man range in length from 90 to about 200 mm., although met with in horses and asses as short as 50 to 60 mm. The question of the differences in length is, however, one depending on the degree of maturity of the worm.

GRASSI'S *F. inermis* measured about 16 cm. long and ·47 mm. broad. The cuticle was 9 μ in thickness, transparent and marked by fine transverse striations throughout the greater part of the length of the worm, but disappearing towards the head and the extreme end of the tail. It presented an outer finely ridged epidermal layer and an inner layer or corium, and below it was visible the coarser longitudinal striae of the dorsal and ventral musculature.

The head was slightly tapering and truncated, devoid of papillæ, with a very small terminal central orifice formed by a continuation of the cuticle into the narrow funnel-shaped pharynx which widened out to form the short œsophagus of ·6 mm. length and thence continued on as the main gut.

The greater part of the cœlom was occupied by the coils of the uterine tubules which were crowded with embryos, some still within the ova, others free, measuring 350 μ in length and 5·5 μ in breadth, with blunted anterior extremity and very thin finely curved tail.

These tubules communicated with a pair of genital tubes which unite a little behind the commencement of the main gut, and find an outlet at the vaginal opening piercing the cuticle at a point ·3 mm. from the oral aperture.

The tail is described as curved with blunt pointed extremity, and the anus, not always visible, as a cleft a short distance above the tip of the tail.

Viewed from the ventral surfaces the end of the tail presented a median, and two lateral pitted markings suggestive of canalicular orifices, but more probably points at which the cœlomic cavity comes into close apposition with the external layer of the cuticle.

GRASSI considered that his specimen differed from that of ADDARIO and, therefore, described it under a new name as belonging to a separate species.

But it is now generally agreed that *F. inermis* is synonymous with *F. conjunctivæ*, as are also BABES' *F. peritonei hominis*, PACE'S[3] *F. palpebralis* obtained from a cyst in the upper eyelid of a Sicilian native of Palermo, in 1887; RIVOLTA'S[5] *F. papillosa*, a mature

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female worm, measuring 5½ cm., found in the eye of an ass, in Pisa, and *F. apapillocephala* of CONDORETTI.[6]

The most recent example of *F. conjunctivæ* was recorded by ALESSANDRINI[2] in 1906.

This case is of particular interest, for the site of worm was almost identical with that in Pte. J. B. (Case I.)

A painless tumour had persisted for 13 months on outer surface of the upper part of the left forearm of a woman, aged 25. Subsequently the arm became painful and swollen, and an abscess which had formed burst, discharging pus and with it the filaria.

ALESSANDRINI suggests that the infection had been conveyed from a horse or an ass through the *Chrysops excutiens* fly.

REMARKS.—The male filaria obtained from the arm of Pte. J. B. was at first regarded as possibly belonging to the species *Onchocerca volvulus* (LEUCKART, 1893), which is well known to occur among the natives of West and Central Africa, giving rise to the formation of subcutaneous nodules in various parts of the body. Native French colonial troops from the Senegal were frequently passing through the hospital, and were thought likely to have provided a possible source of infection through the agency of a mosquito or some biting fly.

The question of geographical distribution, therefore, seemed to have been accounted for, and on clinical grounds also there was some support in favour of this filaria belonging to the Onchocercæ.

Such access as was possible to the literature of onchocerciasis in Salonika shewed, however, that though the worm presented certain resemblances in its fine cuticular striation, and the absence of papillæ from the head, it was of abnormal length for the male *Onchocerca volvulus*, which has not been recorded longer than 35 mm., and its tail shewed none of the typical papillæ presented by that of the *Onchocerca volvulus*; further, it was the only worm present in the tumour. Its identity was, therefore, unsettled.

It was not until my return to England, some months later, that I had an opportunity of consulting Dr. LEIPER, who unhesitatingly pronounced the specimen not to belong to the Onchocercæ, on the following grounds:—

(1) The geographical distribution: *Onchocerca volvulus* has never yet been found in Europe or outside Africa.

(2) The structure of the tumour enveloping the worm is more spongy and much less fibrous than in the nodules of Onchocerca. The latter also invariably contain at least two female and several male worms, and swarm with embryos.[7]

(3) The appearance of the cuticle is quite different, the transverse striations being finer, and there being a total absence of the coarse ridge-like cuticular folds common in the *Onchocerca volvulus*.[8]

(4) The formation of the tail, including the absence of any terminal papillæ, is altogether unlike that of *Onchocerca volvulus*.

Dr. LEIPER has further identified the two specimens as being the male and female types of *F. conjunctivæ* of ADDARIO, the former for the first time on record.

The anatomical structure and appearance of the two specimens bear every resemblance to these species, whose synonyms have already been mentioned.

F. conjunctivæ has hitherto only been found singly, usually in a state of immaturity, within the substance of small spongy fibro-cystic nodules occurring in the neighbourhood of the eye, but not restricted to that region.

So far as at present known its geographical distribution is confined to South-Eastern Europe.

Source of Infection.—Both cases here described occurred in the neighbourhood of horse and mule lines, but the animals not being regarded as possible hosts were not examined.

In elephantiasis (*F. bancrofti*)—possibly also in onchocerciasis, for the two conditions have been found together in a number of instances in Central Africa[9]—the transmitting agent of infection is known to be the *Culex fatigans* and other varieties of mosquito.[10] It is possible, therefore, that transmission of the embryos of *F. conjunctivæ* may be effected

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by a mosquito, or a biting fly, such as a *Chrysops excutiens*, or the stable fly *Stomoxys calcitrans*, the intermediary hosts of *Filaria labiato-papillosa*.[11]

Presence of Microfilariæ in the Blood.—Neither of the patients, though examined both by day and night, shewed any microfilariæ. In the existing records of the few cases of *F. conjunctivæ* no mention has been found as to the condition of the blood. This and the nature of transmission of infection, as to whether the filarial embryo may develop at the site of its introduction, or, gaining the blood or lymph circulation, become lodged in a capillary, and there give rise to the formation of the fibro-cystic tumour, are important points requiring investigation.

Occurrence of other forms of Filariasis in Macedonia.—As the result of inquiries made of several distinguised French and Serbian pathologists, to whom I shewed the male specimen in Salonika, including Professor LISBONNE, M. JOYEUX and Dr. HIRSCHFELDT, I learnt that no case of filariasis at all resembling that of Pte. J. B. had come to light among French or Serbian troops up to June, 1918, also that in spite of the number of native troops from the Senegal no case of onchocerciasis had been reported.

This was also confirmed by M. GAU, Pathologist to the 3rd French Hospital, who was making a special study of the subject of filariasis among native colonial troops from Madagascar, Senegal and Indo-China. He had met with three cases of infection by *Filaria loa*, and in the course of several hundreds of blood examinations had found the embryos of *F. bancrofti* and *F. perstans* in a certain percentage of African natives, who, as "carriers," thus become potential sources of the spread of filarial disease in Macedonia through the transmitting agency of the ubiquitous mosquito. As a result it is very conceivable that the geographical distribution of filariasis may in future embrace regions where infection by *F. bancrofti* and *loa* have been quite unknown.

In conclusion, in addition to my indebtedness to Dr. LEIPER—without whose kindness and wide experience it was impossible to identify the filariæ—I have to express my special thanks to Capt. A. A. LEES, M.C., R.A.M.C., for his help in connection with Case I., and his kind thought in forwarding me the specimen and notes of Case II., to Major MILLS and Capt. McDOUGALL for permission to publish the case, and to Lieut.-Col. WENYON, C.M.G., R.A.M.C., for kindly providing me with sections of the tumour from Case I.

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NOTES AND COMMENTS.

In the paper recently read before the Society by Lieut.-Col. TULL WALSH (Vol. XI., No. 3, TRANSACTIONS) there are one or two omissions in regard to distribution which it might be well to call attention to.

In speaking of pellagra (p. 117) North Africa only is mentioned, whereas it is certainly known in South Africa and Central Africa.

Again (on p. 118) he says, "Ainhum is a very restricted disease, unknown in any part of the world except Eastern Asia and among the negroes in West Africa." This disease is, however, well known in Central Africa (Nyasaland and late German East Africa).

Goundou is also occasionally seen on this side of Africa as well as in the West of Africa.

To give a third instance (p. 119), yaws is very prevalent in many parts of Central Africa, though Africa is not mentioned by Lieut.-Col. TULL WALSH.

On the other hand (p 117), scarlet fever is included in a list of diseases which "are either universal or potentially so." I think many practitioners in the tropics will be inclined to disagree with this dictum, believing that scarlet fever does not occur within certain tropical latitudes.

I should like to add that rickets is unknown in all the Central African races I have seen, as is, I believe, rheumatic fever.

I can also endorse Major F. CHARLESWORTH's remarks in the discussion. No case of blackwater fever in a native in Nyasaland is known, but Indians are frequently attacked by the disease.

HUGH S. STANNUS,

New Langenburg. *Hon. Major.*

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TRANSACTIONS

OF THE

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Proceedings of a Meeting of the Society held on Friday, November 15th, 1918, at 5.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Surgeon-General Sir DAVID BRUCE K.C.B., F.R.S. (*President*), in the Chair.

SANITARY AND INSANITARY MAKESHIFTS IN THE EASTERN WAR AREAS.

BY ANDREW BALFOUR, C.B., C.M.G., M.D., TEMP. LT.-COLONEL R.A.M.C.

Director-in-Chief, Wellcome Bureau of Scientific Research

Necessity is the mother of invention, and war may well be termed the father of necessity, for certain it is that in war man is time and again in dire need. He has to make use of whatever material he can lay hands on. He has to set his wits to work to devise expedients. He has to invent and improvise on the spur of the moment to meet exceptional circumstances, and it must be admitted that his ingenuity is remarkable. If this is true of the combatant, as witness the tanks and hundreds of other marvellous devices, it is equally true, though on a more modest scale, of the Sanitarian. It has occurred to me that it may be of interest to the Society to consider some of the makeshifts of the army hygienist in hot climates. I have been privileged to visit all the important tropical and subtropical theatres of war, sometimes I fear as a kind of stormy petrel, and, thanks to Mr. Wellcome—who placed the resources of his Bureau wholly at the disposal of the War Office—I have been able to secure a large number of instructive photographs, some of which I propose to shew you, and which will serve as the text of a sanitary sermon. I may say that I was a member of the Medical Advisory Committee which visited Egypt, Mudros, Gallipoli, Salonika, Malta and Mesopotamia. I then accompanied Major-General PIKE, C.M.G., D.S.O., A.M.S., to East and South Africa. Finally, at the invitation of General ALLENBY, I went to Palestine to see the anti-mosquito operations there.

Like a sermon, the address is divided into heads for, contrary to the usual army view (I am not referring to the army *medical* view), sanitation is a very large subject, and I have no hesitation in saying it is, or should be, the most important branch of medical

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work in the field. The term "medical," as here used, does not include surgery. It is a trite saying that prevention is better than cure. Everybody knows it, everybody seems to agree with it, but only a minority practise it, and that is why hygiene is, as I have heard it called, the Cinderella of the Medical Services.

Now, in army parlance, sanitation is still most unfortunately almost a synonym for latrines. It looks as if it would take half a century yet to drive this idea out of the minds of a generation which, thanks to faulty initial education, scarcely yet know how they live, move and have their being. It is a sad business, for it spells human lives and untold misery and grief and wretchedness; little crosses in lonely places, wrecked homes and a vast burden of expense and inefficiency.

Still there seems some prospect of better things, and one looks forward to the day when the sanitary flag will alter its hue and sport a mingled blue and white, the white of purity, the blue of hope.

Sewage Collection and Disposal.

For once, however, I will be fashionable, and give pride of place to the subject of sewage collection and disposal, the great importance of which no one will deny, though I would again forcibly protest against the prevalent idea that these duties sum up what is meant by army sanitation.

Different lands, different methods. What may do very well in Salonika is no use in Mesopotamia; what answers admirably in Palestine is hopelessly out of place in East Africa. The latrine very often must be devised to suit the locality. The latrine is, of course, the place of collection, and it may be temporary, or as permanent as anything could be in the kaleidoscopic war which—so far as hostilities are concerned—has just ended in so glorious a fashion. It may or may not be also the place of disposal. When this is *not* the case, there must be some form of special receptacle for the collection of urine and of excrement. There is also, as a rule, for reasons of decency and shelter, a building, and for reasons of comfort a seat, but, believe me, neither of those is a *sine qua non* in war. Let us, however, look for a moment at buildings and seats. We need not worry much about material. Anything may have to serve, from sacking on poles to corrugated iron. In Egypt, matting, or boorsh, as it is called, stretched upon a wooden framework, answered well. In East Africa, grass or reeds served the purpose (*Figs. 1 and 2*); in Mesopotamia, mud was ready for the moulding. One of the chief points about a latrine is that, however makeshift it may be, it should be provided with head-cover. That is not to say it should be completely covered over, but sufficient roof should be provided not only to shield the user from sun and rain, where rain occurs, but to ensure that the receptacle shall be in the shade (*Fig. 2*). There used to be, there still is, a cheerful idea that the tropical sun has merely to glow upon fæces for a few minutes to sterilise them, that nothing evil can live beneath its rays, that, so to speak, sunned fæces are safe fæces. Nothing could be further from the truth. Quite apart from the fact that the sun cannot do these things (you will doubtless remember that in the Punjab it has been shewn that typhoid bacilli can survive 23 hours of a sun drench), fæces with sun playing upon them are those upon which flies, and, at Basra, great brown and yellow hornets swarm and feed. In East Africa especially, I was struck with the contrast, though it is visible everywhere—the shaded latrine had few flies and they were scattered more or less, the sunny latrine swarmed with them and they were concentrated on the pan contents. All latrines therefore should be covered. Where it can be managed the walls should not come right down to the ground. It is in latrine angles that dirt collects, and the angle with the ground is one which can often be avoided (*Fig. 15*). Ventilation is easily secured by leaving a space between the roof and the walls. As a matter of fact most makeshift latrines are only too well ventilated. I have known some which resembled caves of the winds.

And now as to seats. It may be said at once that the less seat the better, for then there is the less to foul. The Indian, the Egyptian and the African native, thanks to their physiologically sound squatting habits, do not require seats at all, a very fortunate provision. The perverse European is fond, however, of providing them with seats.

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What is the result? I recall the story of an ingenious person who made quite a mint of money in the old town of Edinburgh by attracting crowds into a modest building with the following outcry:—

> "A penny, a penny, a penny to see
> A horse with its head where its tail ought to be."

The expectant citizens found only a horse standing with its tail to the manger. Now this was unlikely to lead to anything very insanitary, but it is quite a different matter when one of the Egyptian labour corps carefully supports a falling latrine lid with his forehead, and deliberately ruins the aim and object of a portable fly-proof latrine admirably designed from a sanitary standpoint.

I hope to shew you various types of seats, and will then descant briefly on their faults and virtues. Of special interest is that constructed by the Boche for safari in East Africa (*Fig. 4*). It is in the form of a camp stool with two seats, the upper of a normal type, the lower fashioned as a latrine seat, and therefore possessing a suitable aperture with leather-bound edges. I fear it must be classed under the insanitary makeshifts, though it is ingenious and must add to the traveller's comfort.

We turn now to the receptacle. Rarely, very rarely, has the sanitary bucket, that pride of the Ordnance Department, been visible in large numbers (*Fig. 15*) There is really no need for it, thanks to the petrol tin and the petrol drum. Where we would have been without these aids to hygiene I cannot say. They have played almost as important a part in the war as the tanks.

The Macpherson system, with which you are doubtless all familiar, was much in evidence in Macedonia, and I have also seen it and, I grieve to say, smelt it in France. Theoretically, it is an excellent system. Under rigid supervision it may be a success. Practically it is a failure, human nature being what it is.

To some extent the receptacle must vary with the method of disposal. The pail system can be used either when trenching or incineration is employed; the half petrol can and other small collecting reservoirs are an integral part of the incinerating process. In temperate climates, when incineration is employed, the urine, passed when at stool, is a source of trouble, and must be separated from the solids. This is done sometimes in the tropical war areas but is rarely necessary. The quantity is much less, and with a good incinerator everything goes, as the sanitary corporal aptly puts it, to blazes! Still, at Aboukir, in Egypt, I saw a pail constructed on the principle of a colander, a double pail with the bottom of the inner bucket perforated to allow the liquid to drain away. Its solid contents then went to the fire. Another useful type is the Mackenzie pattern, where one petrol drum is slung inside another.

One of the best and most ingenious methods I have witnessed was in use at a carrier hospital at Dodoma in East Africa (*Fig. 5*). Here the metal vessels in which engineers carry earth formed the receptacles, and when full these latter were picked up by tongs and bodily thrust into the flames. In this way the contents were got rid of, and the receptacles sterilised at one and the same time,

Of all devices the sliding or drawer petrol-can is probably the neatest and best (*Fig. 6*). I shew you pictures of it, and you will see that it has several advantages. It fits close under the seat and cannot get out of place; it hangs suspended, and so the bottom of the tin does not get worn away, and there are handles which keep the sweeper from fouling his hands.

Of the making of incinerators there is no end. Nearly every sanitarian invents an incinerator, and the remarkable thing is that most of them can be made to work. I said "made to work" advisedly, for much more depends on the stoker than on the stokehold. The running of an incinerator is an art, and a good incinerator man should always receive extra pay. Another important point is that, whether he is good or bad, he should live close to the altar which he serves. How many times has one found a latrine neglected because a sweeper was away at lunch! Somehow one forgets that sweepers appreciate lunch like anyone else. Considering their work it seems a little hard to credit, but it is none the less true. To meet occasions of this kind, and also those where a deficiency in

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sweeper personnel prevents prompt clearance of the tins, an assistant-surgeon of the Indian Medical Service in Mesopotamia devised an ingenious and yet simple method of covering the pans and their contents. Not only does it do so efficiently, but it overcomes the difficulty caused by the racial and religious prejudice of many Indian soldiers against touching anything unclean. Two metal hoops like croquet hoops are placed one behind and one in front of the double tins (for urine and fæces) supplied to Indian units. A long piece of sacking, which may be soaked in creosol or arsenite of soda, is attached by one end to the posterior hoop; the other, or free end, in which a piece of wire serves as a handle, being hooked up to the latrine wall. The user, after easing himself, detaches the sacking from the wall and lets it fall over the tins, which it shrouds like a curtain, but, being supported by the hoops, does not touch (*Fig. 7*). I saw this system being used successfully not far from Kut, but it is well to remember that "out of sight" is apt to be "out of mind," hence this scheme should only be employed to meet a special difficulty.

A great deal is said about the necessity of incinerators being provided with baffle-plates and combustion chambers. Where very large numbers of men have to be catered for, and where the vicinity is populous, this is, no doubt, necessary and desirable, but hundreds of incinerators which never possessed baffle-plates are doing good work to-day. I cannot describe them all at length, but I exhibit photographs of different types (*Figs. 8 and 11*), and direct attention to ingenious makeshifts, especially the tin and mud open "cone" incinerator for horse manure, as used in Mesopotamia, where, remember, there is practically no stone and plenty of scope for the inventor (*Figs. 9 and 10*).

One thing I would like to say: If at all possible, place your ordinary incinerator on a cement foundation (*Fig. 11*). Otherwise the ashpit is very apt to become a fly nursery. In Mesopotamia the number of fly maggots found living cosy if precarious lives under incinerators was amazing.

The motto of all who incinerate must ever be "from the pan direct to the fire," otherwise incineration may become a very dangerous method of disposal. The sweeper who mixes bhusa and fæces on the ground, and leaves the mass to the flies until he feels inclined to shovel it piecemeal into the fire, is only too common, and is a danger to the Forces (*Fig. 12*).

In Mudros I got one of Lumsden's home septic tanks—with which you are doubtless familiar—erected, and it worked quite well. I did not see another till I was at Potchefstroom, and there, at a demonstration centre, I obtained the photograph which I exhibit (*Fig. 13*). This is a cleanly method, and I understand that farmers in the Transvaal are now adopting it. As you know, it was originally introduced for the hookworm campaign in the Southern States of America.

And now a word as to latrines without receptacles.

The most familiar is, of course, the box-seat type with falling lids, placed over a deep trench. It has been in use on all the fronts I have visited, and one may say of it that when it is good it is very, very good, and when it is not it is horrid. In other words, when properly constructed, properly looked after and properly used it is an excellent sanitary contrivance. Too often, however, one or other of these desiderata is lacking, and then the trench below it becomes one vast breeding ground for flies. This is what happened in the Sinai Peninsula. The box-seats cracked and warped with the sun; they were not repaired, and flies soon found their way through cracks and holes and broken lids, attracted by the irresistible odour of the trench contents. To some extent this difficulty can be got over by constructing the box partly of corrugated iron, as was done in Macedonia (*Fig. 14*), but I fear the box latrine has not fulfilled the hopes of its admirers. There is, however, a modification of the system which has much to commend it. Set at intervals over a long deep trench, supported on iron rails, and provided with rims and covers or little box-seats with falling lids, are set a number of petrol drums, which serve as pedestal shafts to the trench (*Fig. 3*). The latter is roofed in elsewhere and covered by beaten-down earth. Such a system certainly lessens the risk of fly infection, but, to make assurance doubly sure, a certain German doctor long ago invented the smoke method, and in many parts of E. Africa one encountered the smoke latrine. The principle is simple, and is shewn in the photo. (*Fig. 22, smoke urinal*) and drawings. Here, again, however, care and supervision are

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necessary. I remember a certain officer commanding a hospital, who led me with pride to see his smoke latrine. The lid was removed, the smouldering mass of leaves and wood, evolving pungent fumes, was lowered in the brazier, and immediately, with a loud and angry buzz, there emerged green Lucilia, grey Sarcophagidæ, blue Calliphora, swarms of small Muscidæ, and all the flies which should not have been there but which, owing to neglect, had got a chance of gathering to the feast.

The river Euphrates and its fish played the part of receptacles at Chabaish in Mesopotamia (*Fig. 16*).

Units on the march have no time for elaborate latrines. They have to employ comparatively shallow trenches. Now, if these are not protected at the time of use, and not properly filled up and treated after use, other units following after will be plagued with flies and possibly with dysentery. Hence the value of what may be called the portable-plank method. Each unit should carry these creosoted or cresoled planks, of which you will note the dimensions, and use them as temporary trench covers, alternate planks being removed, and the men straddling across the gaps and replacing the planks (*Fig. 17*). A guard must be posted to see the operation is properly conducted. Full details are given in the "Memoranda on War Diseases" issued by the War Office.

Lastly, I shew you a naval improvisation, the type of latrine for native troops, adopted after much tribulation on the Victoria Nyanza steamers. It is merely a framework, covered with cloth or tarpaulin, projecting from a port (*Fig. 18*). It effectually prevents fouling of the vessel's sides.

A few other points and we turn to a more savoury subject. As you know, the Indian practises "abdust" or lavage, and it is very important that he should do so under sanitary conditions Here is the best scheme of many that I have seen (*Fig. 19*). A square pit is dug and filled nearly to the surface with tins or rubble. It is then covered over, but a hole is left on each of its four sides. Sloping channels, lined with tin, lead to each of these holes, and the Indian, straddling across the channel, performs his ritual *sans peur et sans reproche*.

The question of field urinals need not detain us. They vary from the ubiquitous funnel—at first mistaken for a listening-post (*Fig. 20*)—to the East African ant-hill, which has most marvellous absorptive powers (*Fig. 21*). A smoke urinal has also been employed —rather an unnecessary complication (*Fig. 22*).

Food and Cooking.

I suppose it seems heresy to say so, but I am convinced that the excellent health of the troops on the Western Front is quite as much due to the indefatigable efforts of the Army Service Corps as to those of the Royal Army Medical Corps, speaking for the moment merely as regards the question of transport and distribution of food. I am sure that many officers of the Medical Corps will agree with me It stands to reason. Feed a man well—that is, give him plenty of good food in sufficient variety, adapted to his racial peculiarities. to the climatic conditions under which he is serving, and well cooked, and in the great majority of cases he will defy not only the Boche but the bacillus. I am certain also that, anti-mosquito precautions and preventive inoculation excepted, good food and proper cooking are more important sanitary measures than any others in the field. Where were they not forthcoming? In Gallipoli, Mesopotamia and East Africa. What war areas have had the greatest disease incidence, the highest mortality? Gallipoli, East Africa and Mesopotamia. To this day it is not fully realised that questions of food and cooking are scientific questions—that the advice of dietetic experts should invariably be sought and followed. If this were done, one would not see ration scales for native African troops devoid of substances essential to life and health—one dietary without sugar, another minus vegetables. It seems scarcely credible, but it is true. Now cookhouses are important places. In the earlier stages of the war everyone tried to make cookhouses flyproof, with the result of converting them into flytraps. Anyone who was at Mudros will confirm this statement. It is no use employing wire netting in a cookhouse unless you have double doors, can execute instant repairs, and can post a sentry to shoot those who put stones under the doors to keep them open. No, a cookhouse must be well ventilated, as dark as possible, and its door should face north in the northern hemisphere and south if it is on the other side of the equator. All this sounds trivial, but at Lindi, in East Africa I saw

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a cookhouse on which many pounds had been spent, and it was worse than useless. On the other hand, I shew you a fenestrated mud-brick cookhouse, of most excellent design, which I saw near Jaffa (*Fig. 23*). It is a new and original pattern, well worthy of adoption, designed by Lieut.-Colonel OTWAY, R.A.M.C.

The photographs shew you all kinds of open-air kitchens and field ovens. Much ingenuity is often displayed in their construction. The same is true of field-bakeries—most important institutions in which very often wonderfully good bread is baked. Believe me, good bread often means good health, for, as you know, bread is the "staff of life."

I direct your attention to the plan of an underground larder used by a crack cavalry regiment in Mesopotamia (*Fig. 24*). While their comrades spoiled their teeth and their tempers on tough meat, this unit enjoyed tender roasts and stews throughout the fiery heat of a Mesopotamian summer—a fitting reward of enterprise.

Professor SIMPSON, at the last meeting of the Society, paid a tribute to our gallant allies, the Serbians. I saw a good deal of these fine peasant soldiers just after they reached Salonika from Corfu. We had a great deal to learn from such veteran campaigners. While our men too often "pigged it" in their tents—many units at that time having no mess marquees or huts—the Serbians dined in comfort at stone tables under an awning of tree branches, which they collected from a place eight miles away (*Fig. 25*). You will see their stone serving tables (*Fig. 25*), their kitchens, and the officers' dining-table and seats of wicker or basket work (*Fig. 26*), both comfortable and picturesque.

WATER SUPPLIES AND WATER PURIFICATION.

Lack of space prevents any reference to the hundred and one contrivances for dealing with sullage water in hot countries, but the photographs shewn speak for themselves. Of more importance is the question of water supplies and of water purification. The sources of supply varied greatly on various fronts. Egyptian filtered water, brought by tank steamers, was the chief supply in Gallipoli; in Salonika, mountain streams were often a source; in Palestine, the sources varied from the beautiful "Springs of the Sultan," near Jericho, to the clear waters of the rapid Auja river, which flows into the sea near Jaffa, and included the new reservoir supply for Jerusalem, gathered from the hills about Bethlehem. In Egypt, the Nile and its canals furnished the supply, and brought risks of bilharzia; in Mesopotamia, a dry and thirsty land, the Shatt-el-Arab, the Tigris, the Euphrates, the Karun, and surface wells yielded their waters, sometimes with the cholera vibrio in them; in East Africa, men drank from rivers and from water-holes, from mountain streams and from wells, and ran many risks in consequence, for the makeshifts devised for protection were too often well worthy of the name. Let us look at some of them and at methods of collecting, transporting, storing and purifying water on various fronts. Putting a strand or two of barbed wire round a water-hole, and placing a native sentry on guard over it, are measures of slight futility, though better than nothing. Where possible, a high fence should surround the spring or pool, with a hole in it through which the collecting hose passes This was done at Summit, on the Mikese-Rufiji road, in East Africa, and answered excellently (*Fig. 27*). At Amara, in Mesopotamia, the admirably protected well areas with covered wells were one of the sanitary features of the place, and a tribute to the Royal Engineers.

The only special device for collection which I will mention is the use of the huge fruits of the baobab tree as pitchers when drawing water from wells. Methods of pumping, more or less elaborate, are shewn on the screen.

Storage is often a troublesome business, especially in a country like Mesopotamia, where for a long time transport was a great difficulty. That is one reason why large rubber canvas troughs, each holding 2,300 gallons, came into use (*Fig. 28*). Their only drawback was that they were not covered. Tanks hollowed out in the soil, lined by tarpaulin and covered with canvas, were common in the Advanced Corps Area, in 1916. I have seen drinking-water stored in a German shell-case, which makes an excellent receptacle. Unfortunately someone had placed the long metal cylinder in the blazing sun and the water was scarcely refreshing. Even makeshifts must be properly used. A very bad type of watertrough was the canvas diggie made in Basra—a clumsy, heavy, leaking arrangement.

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Water was transported in many ways: by carts, in pakhals and fantasses, on mules and camels, in bags, in petrol-tins as at Anzac (*Fig. 29*)—a weary business, but I do not recall any very novel devices, save the use of iron telegraph poles as pipes at Sheikh Saad in Mesopotamia, and of split date-palm trunks as water-gutters.

As regards purification. In some places I have seen river water boiled in petrol-tins on the bank, and all the good of the boiling vitiated by the transport people plugging the openings in the tins with bunches of grass or reeds—a very insanitary makeshift (*Fig. 30*). On the other hand, an Indian hospital had an admirable arrangement whereby water was sterilised in special boilers and gravitated by tin channels to metal receptacles (*Fig. 31*). At Port Amelia, in Portuguese territory, a British hospital adopted the principle, but placed the receptacles up-hill from the boilers, and so the boiled water had to be carried up to them by hand, an element of risk being thereby introduced. As has been truly said, common sense is rare sense.

Two makeshifts for preparing good emulsions of chlorinated lime are worth noting, both employed in Basra. The earlier one consisted in Indians violently shaking Winchester quarts filled with the solution (*Fig. 32*); the latter was represented by ingenious hand-churns, and worked very well (*Fig. 33*).

Elaborate improvised labyrinths were at one time in vogue in the Suez Canal zone to ensure a thorough mixing of water with alum, and thereby a satisfactory precipitation prior to filtration. The design reminded one of the maze at Hampton Court. A sergeant-major at Amara effectually prevented thirsty souls from drinking out of a tin dipper, and thereby possibly contaminating a water supply, by serrating the edge of the tin (*Fig. 34*); while a good method of cooling water employed in Macedonia was to surround a water-tank with sandbags, which were kept wet, an air space being left between the bags and the tank.

Bathing and Washing.

Great ingenuity was often shewn in the construction of shower-baths, but nowhere more so than in the Corps Area in Mesopotamia, where the absence of wood and stone resulted in the remarkable tin structures, a picture of which I shew you (*Fig. 35*). The simplest makeshift, however, was at Morogoro, in East Africa, where by hollowing out the ground a stream was made to cascade and thereby supply a splendid douche (*Fig 36*). The damming up of natural watercourses often resulted in the provision of excellent baths (*Fig. 37*), and a fine swimming-pool was created by the use of a huge metal tank employed in the soda works at Wady Natrun in the western Egyptian desert. The ordinary ablution bench was very unsatisfactory until one genius thought of adding a rail, on which coats and shirts could be hung (*Fig. 38*). Previously these garments, like "Uncle Pete's hat," had been hung on the ground, and were very apt to get dirty and wet. Talking of cleaning, the plan of scrubbing steamers' decks on the Victoria Nyanza with half cocoa-nut shells came as a revelation to me, and is probably new to most of you.

Disinfection is closely allied to bathing and includes the destruction of vermin. The simplest makeshift for dealing with clothes lice is probably the oldest—the picking method—carried out either by the sufferer or a friend. I shew photographs illustrating both operations, the first taken in the front line trench at Anzac, where the picker was much more interested in his prey than in bullets flying overhead (*Fig. 39*); the second secured in Amara, and rather reminiscent of monkeys at the Zoo (*Fig. 40*). But we can do better than this in the army, and you will find illustrations varying from the Serbian barrel to the most useful railway van disinfector, which of all methods I have seen accomplishes most in the shortest time, as you will find if you peruse Col. Wm. Hunter's articles in the *Lancet* for Sept. 14 and 21, 1918. Both these useful methods owe their introduction to Lieut.-Col. Stammers, R.A.M.C. Now the Serbian barrel is often wrongly used. What is the use of placing it on a hillock and having to climb up to it every time soldiers' kits have to be popped into it (*Fig. 41*)? Again, why do so many people neglect to see that the water-tank, the source of steam, is so placed below the barrel that the largest available evaporating surface is provided? Success in sanitation depends largely on attention to detail, and very often this is not forthcoming.

The so-called Levisseuse apparatus, which is a kind of metal Serbian barrel, has such a limited holding capacity that it is not much use.

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A barrel may be used for holding disinfectant outside a latrine. It should be accompanied by a nail brush fastened to a wire. One medical officer actually arranged a latrine in such a way that once in it you could not emerge without dipping your hands in a disinfectant solution which effectually camouflaged the latch of the door. I fear the plan was more ingenious than effective.

A very insanitary makeshift for getting rid of dead animals on the road was too often in vogue in East Africa, namely, leaving them there till the flies and the vultures made an end of them. I show you the picture of a carcase where, it is true, an effort at charring had been made, but in which the main disinfection was that whereby Herod Agrippa ceased to be a nuisance, for this dead horse was being "eaten up of worms," or rather by the maggots of Pycnosoma flies.

Fly Destruction.

I do not like to call any invention of my own a makeshift, but would like to direct your attention to the open-air cage fly-trap (*Fig. 42*) and to its modification by Capt. Rose. Both these models work well when properly baited and operated. This is not the case when medical officers construct them so as to catch sparrows as well as flies, or alter them according to views which effectively ruin their efficiency, or pay no attention to them after the first excitement of trapping several thousand flies is over. Here are the rules which must be followed if these traps are to be a success: –

1. The calico or canvas forming the greater part of the sides of the trap should be stretched *tightly* on the framework.
2. The slit between the wire mesh and the alighting board should be just large enough to allow a bluebottle fly to enter the trap.
3. The trap should, as a rule, be placed in the sun out in the open.
4. It should be placed outside a mess or cookhouse, or near a latrine, or between an occupied tent or camp and any source of flies.
5. It is essential that it should be properly looked after, especially as regards baiting.
6. For this purpose it is best placed under the charge of some one person who will be responsible for keeping it in repair, baiting it properly, and cleaning it.
7. Any attractive bait may be employed, but one of the very best is chicken entrails. Human fæces may be used if placed in a tin carefully covered with wire gauze, so that the flies cannot get access to the contents. Other baits are:—raw meat, cheese paste, stale beer, bread soaked in vinegar water, jam, marmalade, lentil paste, with a little sugar sprinkled on it, old melon rinds, fruit and fish refuse.
8. Flies in the trap are best killed by having a poison solution such as formalin 3 per cent., made up with lime water, arsenite of soda syrup, or sodium fluoride.

   Whatever poison is used it is best placed in a covered tin, through the lid of which wicks protrude. These wicks dip down into the fluid or syrup which they suck up, and the flies imbibing the poison from them speedily die.
9. Failing poison, the flies can be killed by spraying with spray oil or some similar preparation, or by fumigation, the trap being covered over with cloths or blankets during the fumigating process.
10. A record should be kept of the daily catch. This may be done by weighing the flies or seeing how many will go into a receptacle of known capacity. A pint measure holds about 10,000.

It is perhaps news to many that a tangle-foot fly paper is much more efficient if used in the form of an arch; sticky surface of course upwards (*Fig. 43*). This has probably something to do with the way the light falls upon it, and an apparatus has been devised for holding such curved papers.

The roller towel method of using arsenite of soda solution is an excellent makeshift arrangement, and, with a minimum of attention, ensures a constantly damp, poisoned surface for the delectation of our enemy the fly (*Fig. 44*)

At Nasiriyeh, on the Euphrates, Major Patten, I.M.S., had introduced Roubaud's

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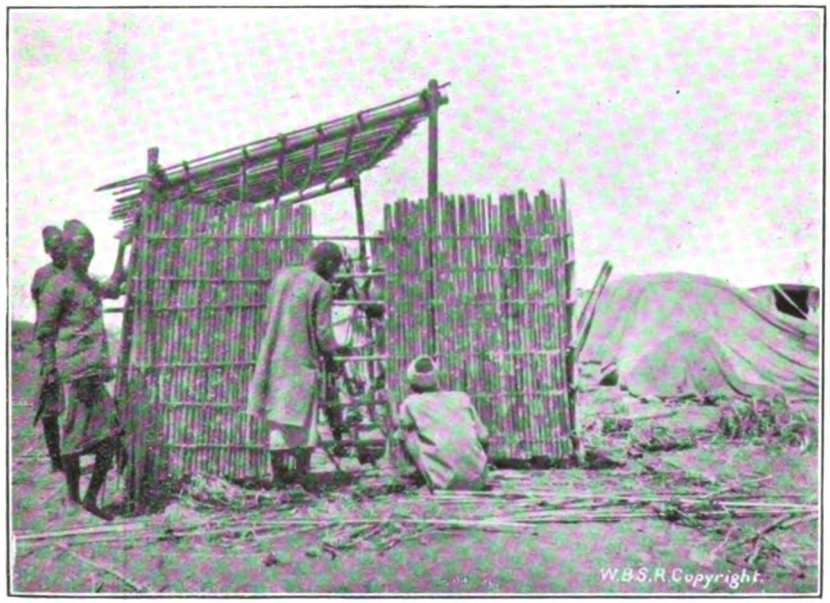
1.—Native Latrine (uncovered), K.A.R. Lines, Tabora.



2.—Latrine for Dysentery cases in process of construction, Port Amelia.

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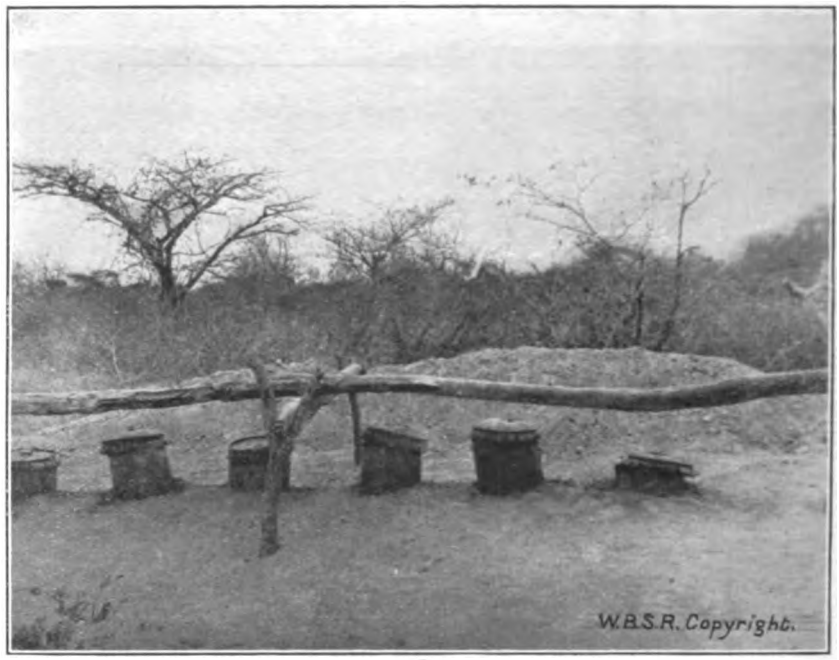

3.—Bad type of trench latrine, Dodoma. The seat is uncomfortable and too high, there is no roof, and the covers of the cylinders leading to the pit are defective.

4.—Adaptation by the Germans of Camp-stool for use as Latrine Seat.

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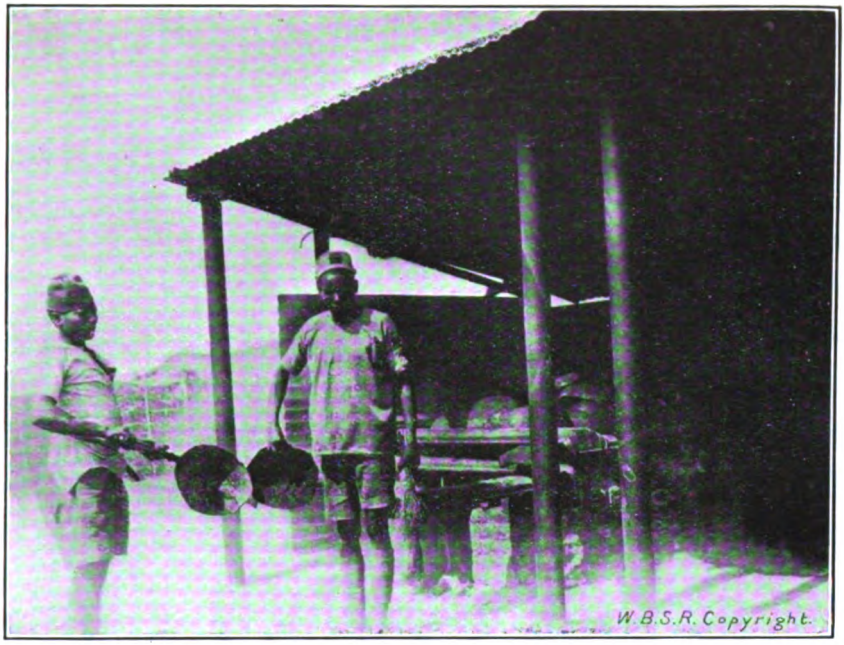
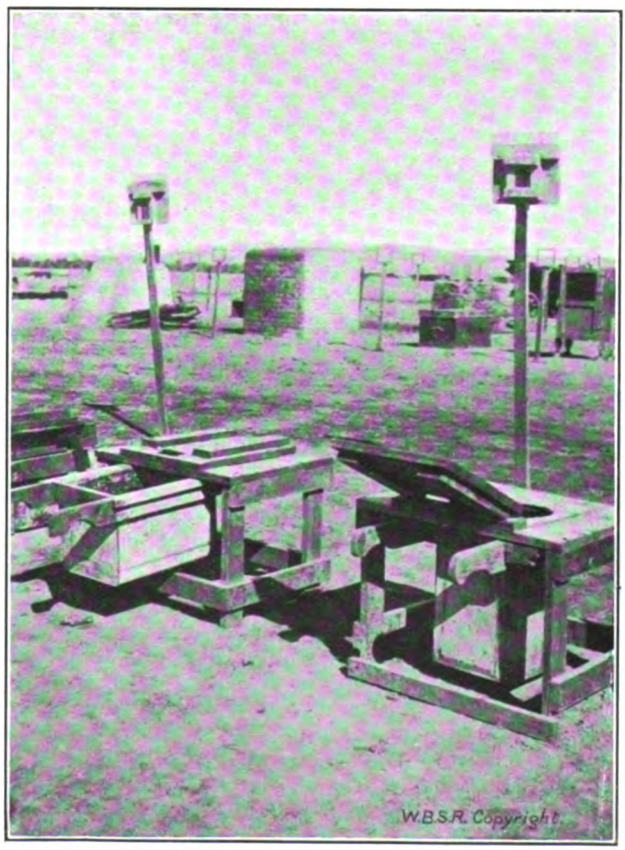


5.—Method of Incineration practised at Carrier Hospital, Dodoma. The fæcal receptacles held by tongs are plunged bodily into the fire. These metal receptacles are those used by engineers for carrying earth.



6.—Sanitary Demonstration Centre, Basra, shewing in foreground types of fly-proof Latrines with petrol tins suspended from sliding runners. The seat-cover on the right is badly arranged. It falls back too far to be self-closing.

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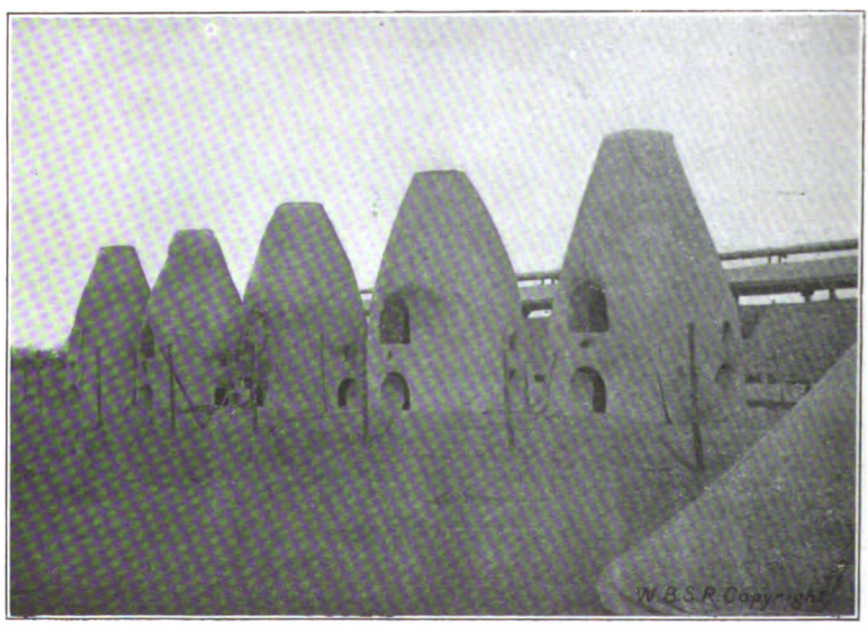
7.—Method of protecting pan contents in Indian Latrine from flies, as shewn at Sanitary Demonstration Centre, Dar-es-Salaam.



8.—Battery of Sugar-loaf Incinerators, Amara. Note proximity to latrine.

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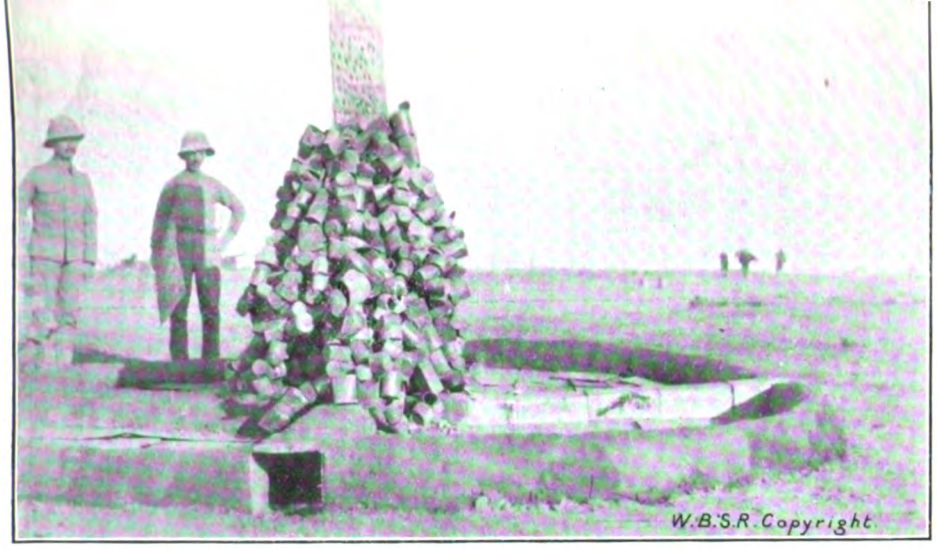
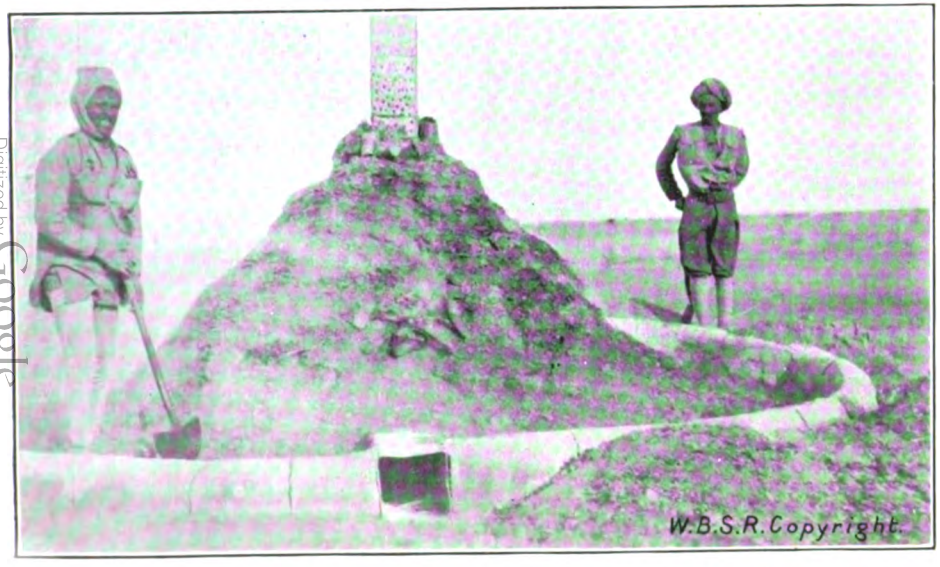
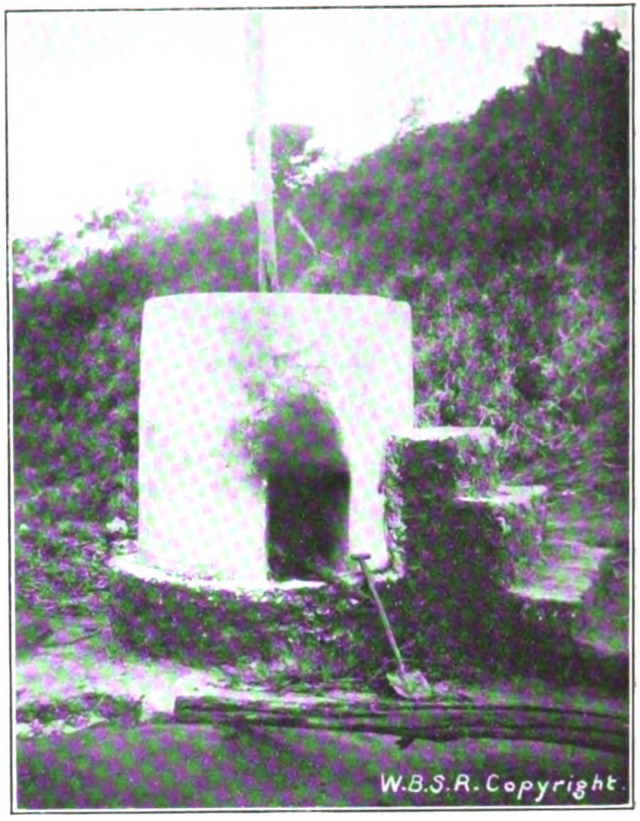


9.—Open Cone Incinerator for House Manure. Constructed of petrol cans, small tins, wire and mud.



10.—Open Cone Incinerator being used for the burning of manure.

Photographs 9 and 10 by Captain P. F. GOW, D.S.O., I.M.S.



11.—Incinerator placed on Cement Base to prevent fly-breeding. 3rd S.A. Field Ambulance, Summit, East Africa.

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12.—Mud-brick Incinerator, river post near Amara. The sweeper is shewn mixing bhusa and human excreta. The mass is often left for a considerable time before being fed to the Incinerator, and in this way flies gain access to it.

13.—Improvised Lumsden Wet Privy or home septic-tank method for dealing with excreta. Note fly-proof seat, closet barrel and effluent barrel. There is an anti-splash arrangement inside the closet barrel. Sanitary Demonstration Centre, Potchefstroom. Urine funnels seen in background.

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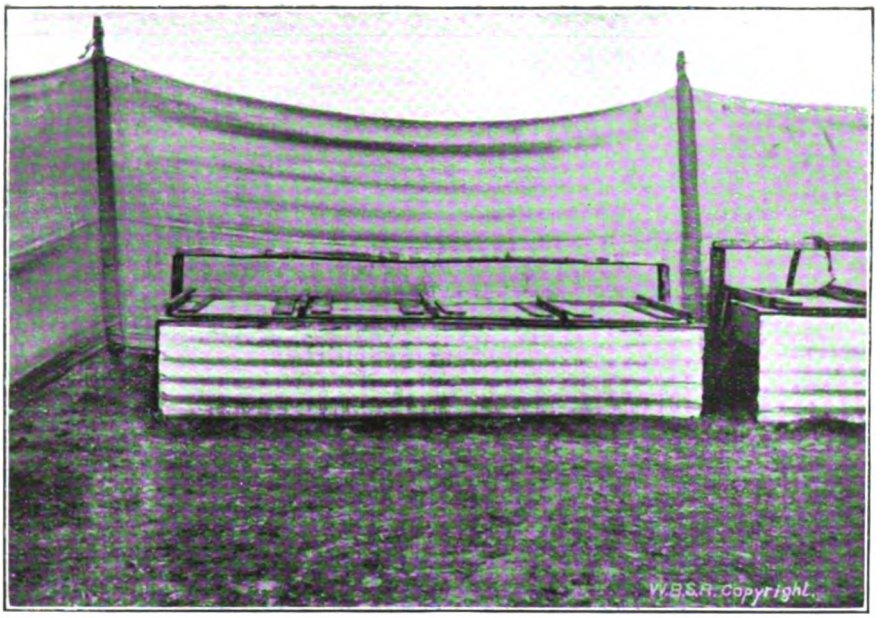

14.—Fly-proof and Wind-proof Box-seat Latrine, partly made of Corrugated Iron.

15.—Good type of Fly-proof Latrine, Australian Camp, Tel-el-Kebir. Separate fly-proof seat with falling lid on each pail. Note litter at the angle formed by the ground and the wall of the latrine..

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16.—Latrines for British Units built out over a creek, Chabaish, Euphrates.

17.—Temporary Latrine covered by Oiled Boards set side by side. This form was employed owing to proximity of strongly brackish subsoil water to ground level, Kantara, Canal Area.

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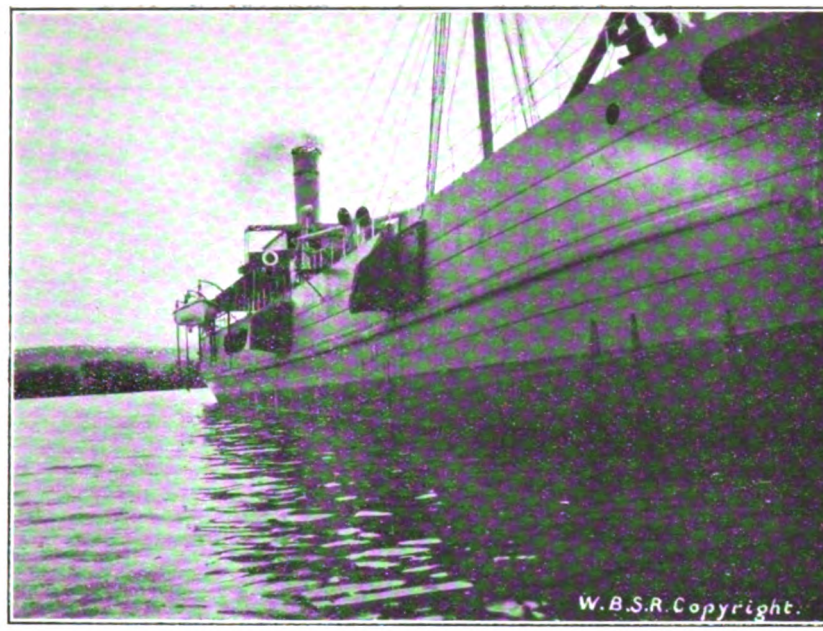
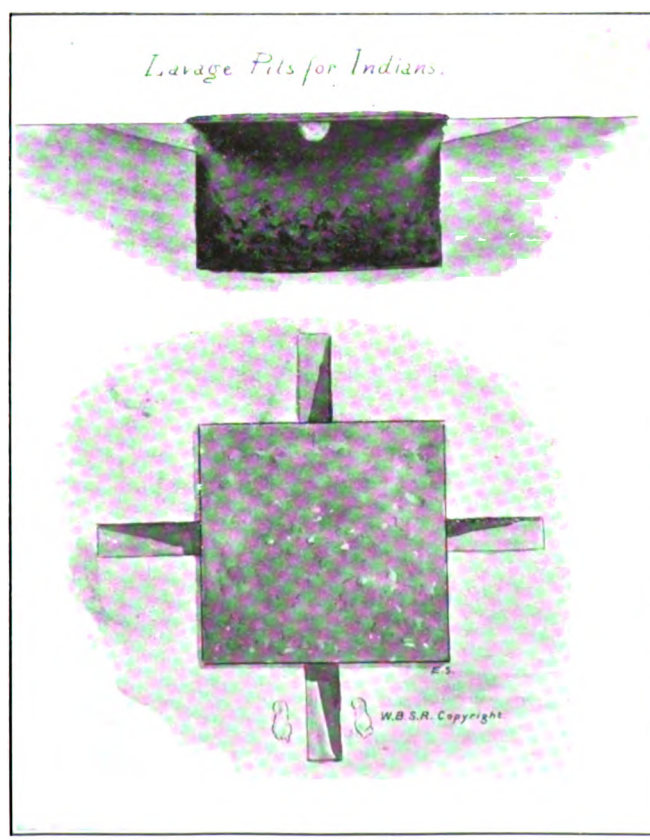


18.—The s.s. "Usoga" on Victoria Nyanza, shewing Latrine arrangement for native troops.



19.—Good type of Covered Lavage Pit for the use of Indians. Note channels which are lined with tin.

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20.—Urinals communicating with soak-pit. One shews flap to prevent dripping on ground.

21.—Large Ant-hill used as Urinal, Dar-es-Salaam.

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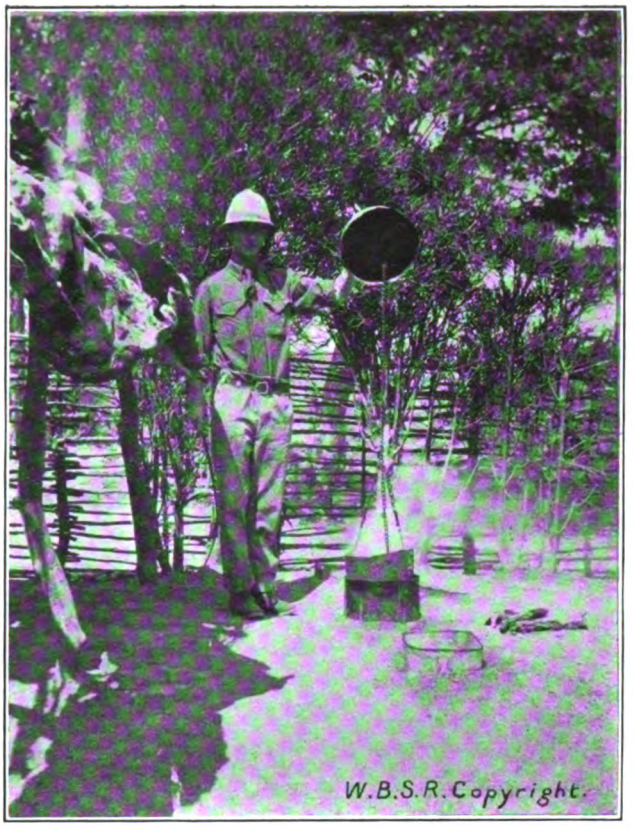
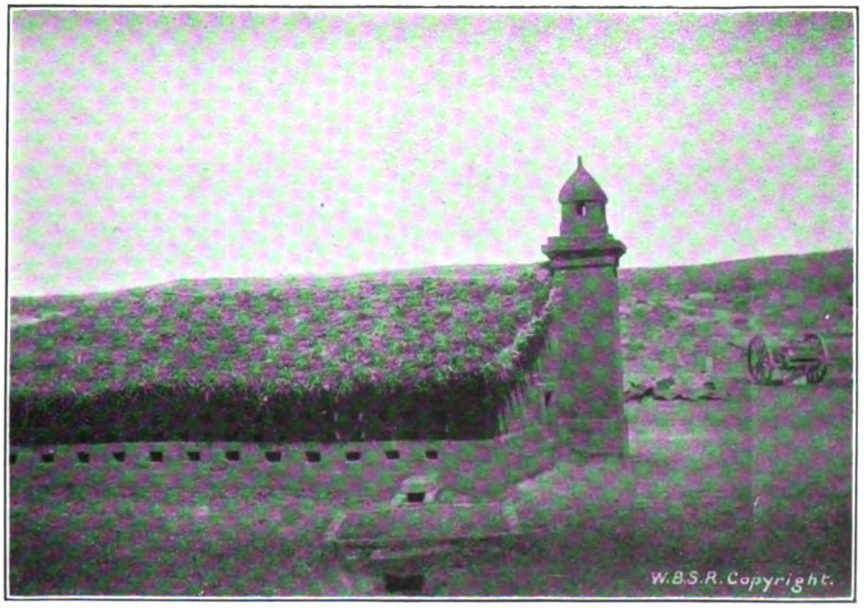


22.—Smoke Urinal, No. 1 Carrier Hospital, Iringa, shewing method of lowering brazier.



23.—Fenestrated mud-brick Cook-house of No. 130 Combined Field Ambulance, on the road north of Jaffa. (Designed by Lieut.-Col. OTWAY, R.A.M.C.)

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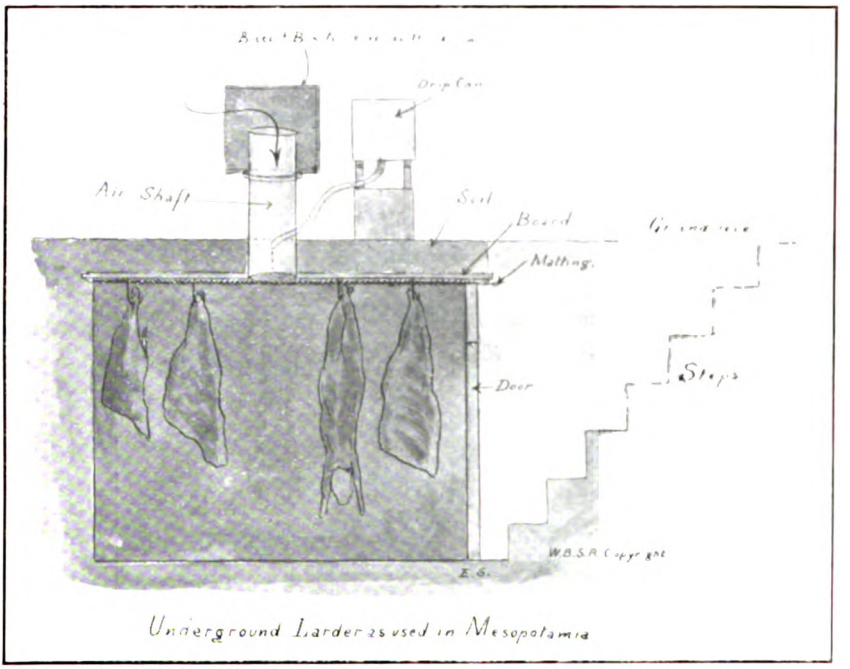

24.—Method of keeping meat, and ensuring that it was tender, employed by a Cavalry Regiment in Mesopotamia.

25.—Improvised Messing Area. Dining-tables and serving-tables made of stone. Roofed with leaf shelter. Serbian Army.

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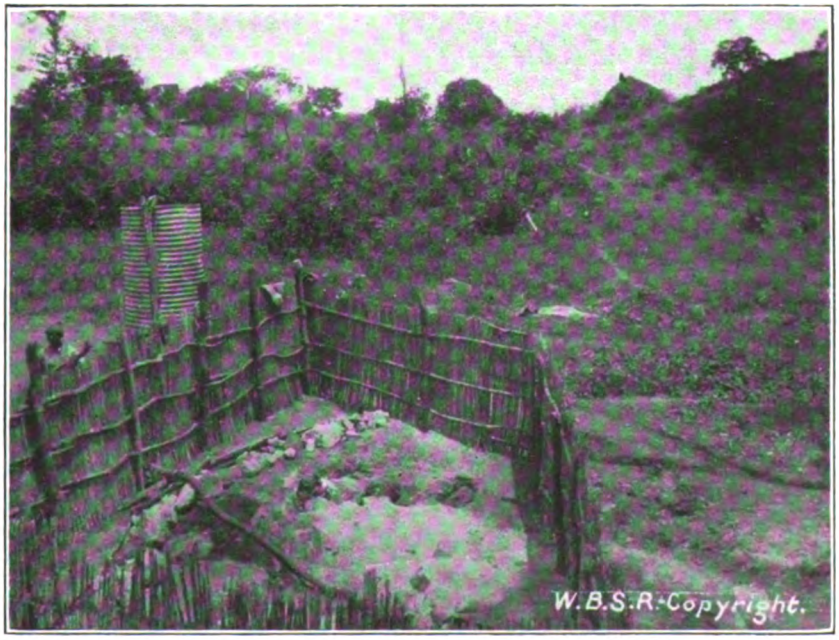
26.—Improvised Dining-table and Seats for officers of Serbian Army. Made of wicker-work.



27.—Well-protected "spring" water supply at Summit, East Africa, shewing hose leading to water-tank.

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28.—Rubber Canvas for holding Drinking Water. These canvas tanks hold 2,300 gallons.

29.—Water Transport in Petrol Tins. Note small Turkish well. Clark's Gully, Anzac. The trenches are on the top of the hills seen in the background.

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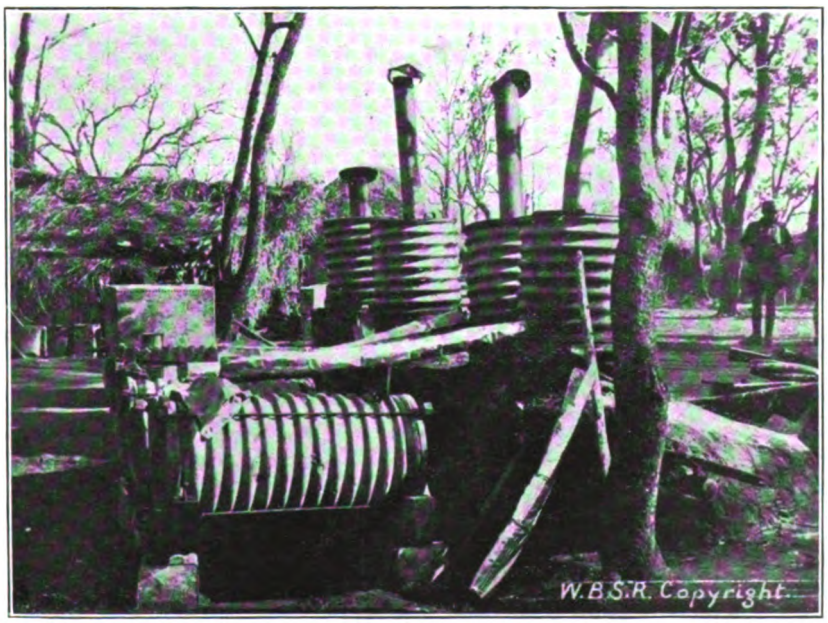
30.—Unguarded Water Supply, River Mbemkuru, Nahungu, shewing collection in petrol tins in which water is boiled on the spot.



31.—Water Boiling Apparatus, Indian Clearing Hospital, Nahungu.

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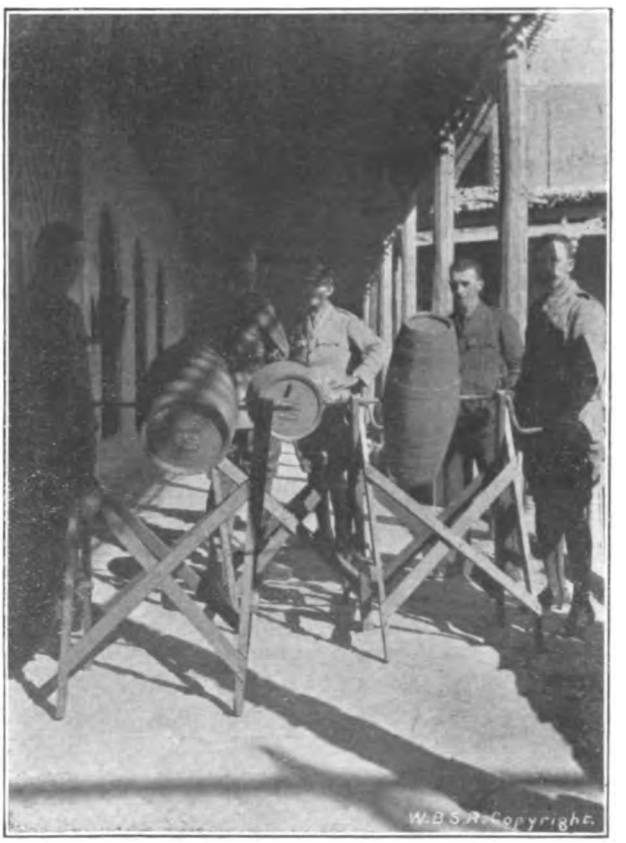
32.—Old Method of Preparing Chlorinated Lime Solution for Water Sterilisation. Indian orderlies shaking bottles. Basra.



33.—New Method of Preparing Chlorinated Lime Solution for Water Sterilisation by Use of Churn.

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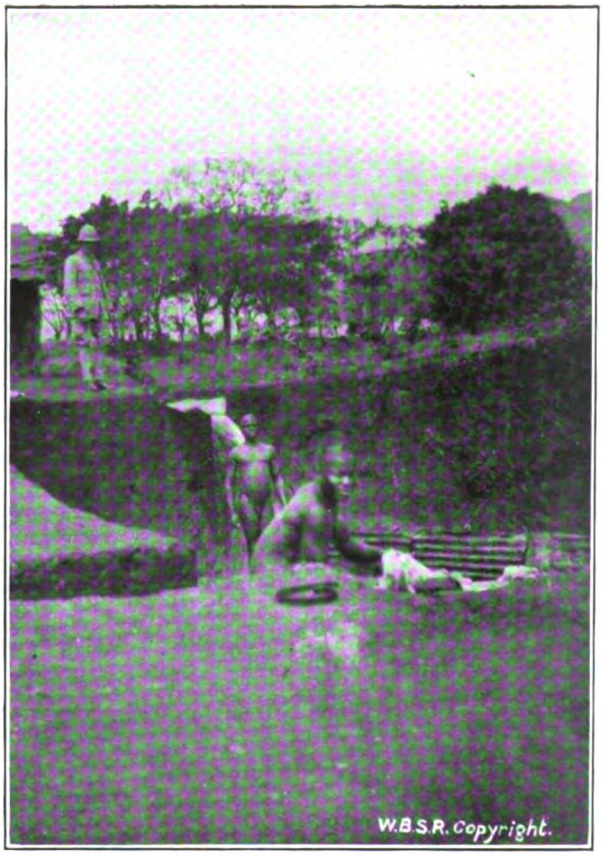
Persian Hub and Dipper, shewing method of serrating the edge of the latter to prevent men using it as a drinking-vessel. No. 23 British Stationary Hospital, Amara.



36.—Improvised arrangement for native ablution, No. 15 Stationary Hospital, Morogoro.

35.—An improvised Shower-bath made from petrol-cans and wire. Highland Nullah, Tigris Front.

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37.—Improvised Bathing-place, Macedonia, formed by damming up the course of a mountain stream.

38.—Ablution Bench furnished with rail for hanging clothes.

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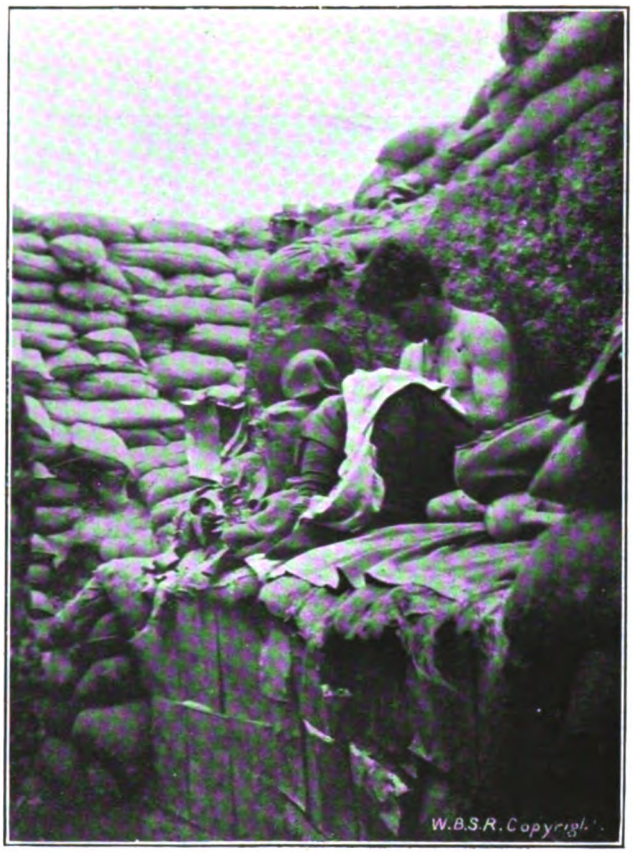
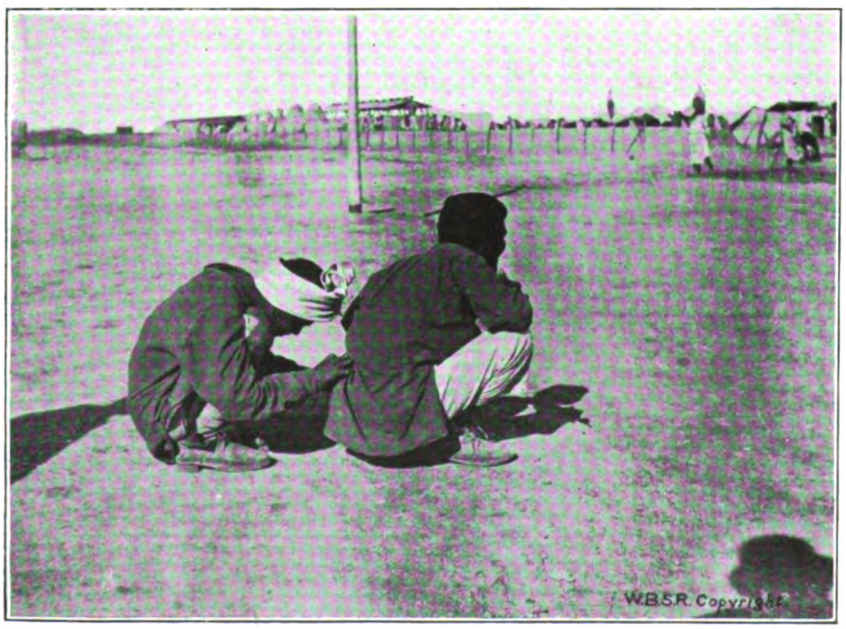


39.—Primitive method of removing Lice. Front line trench, Anzac.



40.—A simple method of Lousing employed by Indian troops, Amara.

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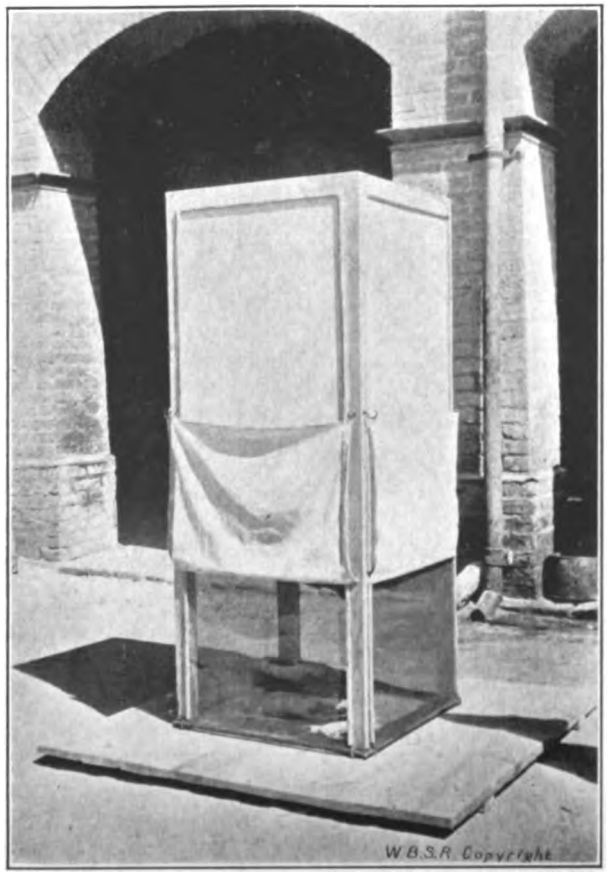
41.—Serbian barrel wrongly placed, making it difficult to insert kit in the barrel owing to height above ground level.



42.—Latest type of Cage Fly-trap, in use at Basra.

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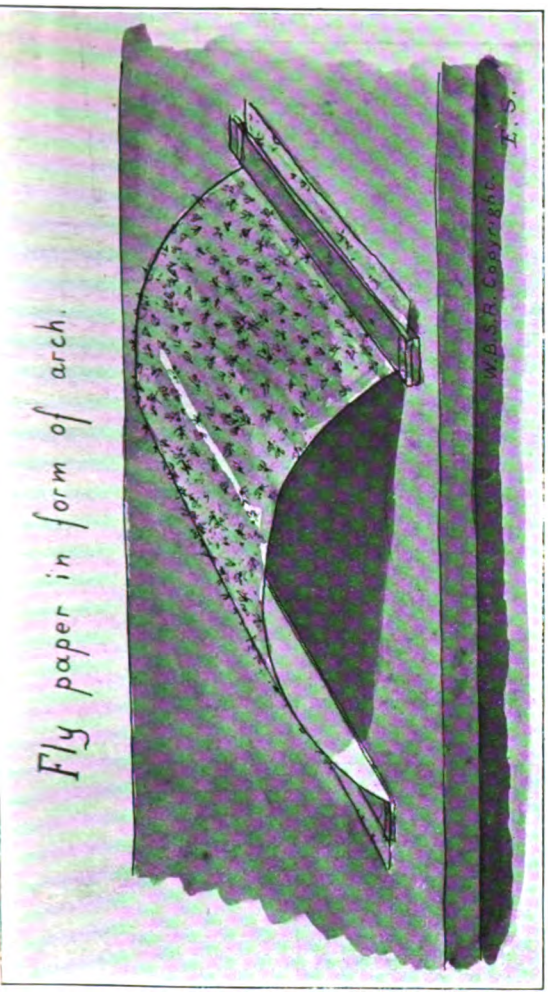
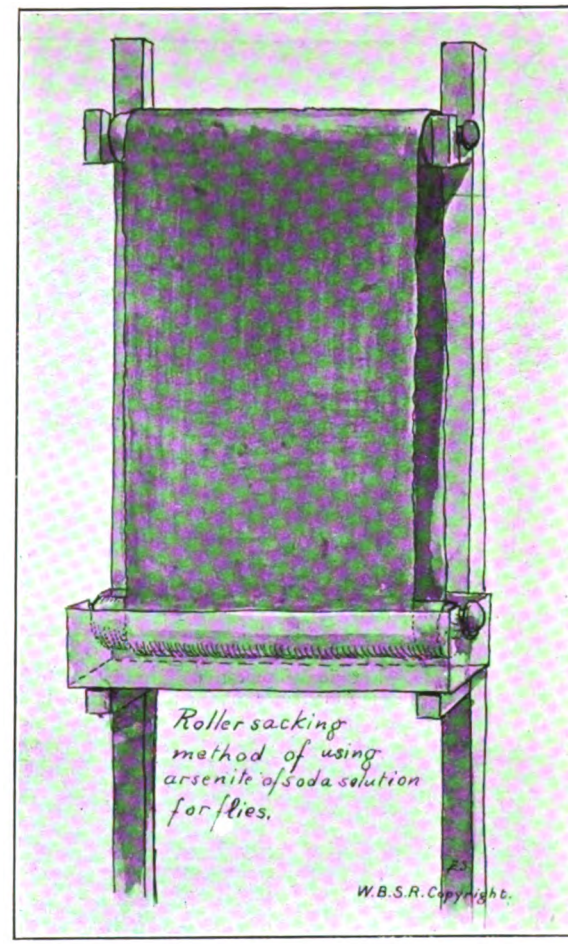


43.—This arched form of Fly-trap is found to serve as a more efficient trap.



44.—The Trough in which the sacking dips contains the poisoned solution.

45.—ROUBAUD's method for the Destruction of Fly Eggs and Larvæ, shewing mounds of manure. Grid and other Incinerators are also in evidence.

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46.—Exterior of Fenestrated Mud-brick Mosquito and Fly-protected Ward, of No. 130 Combined Field Ambulance, on the road north of Jaffa. Designed by Lieut.-Col. OTWAY, R.A.M.C.

47.—Part of 124th Indian Cavalry Field Ambulance, Jordan Valley, shewing Sick under Grape-vine in Monastery.

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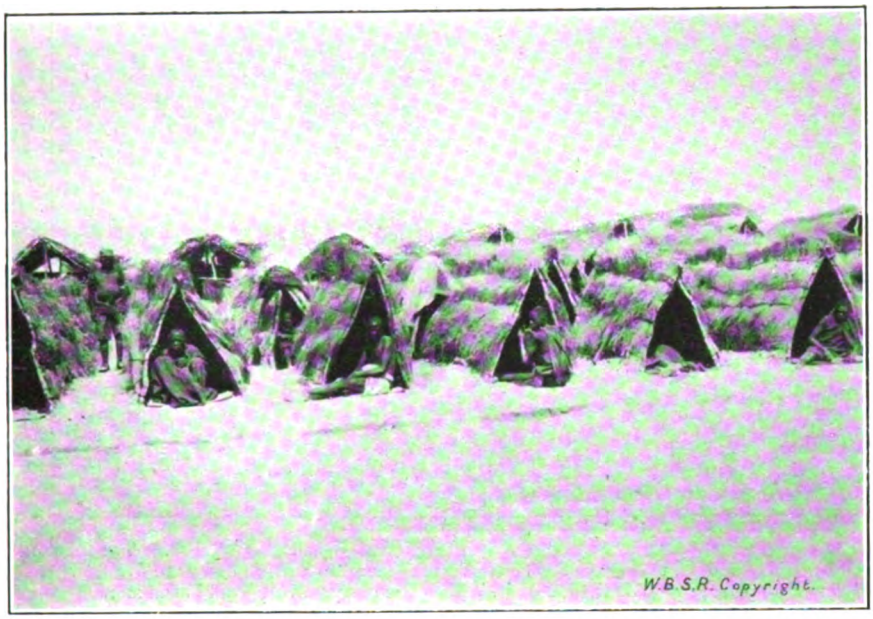
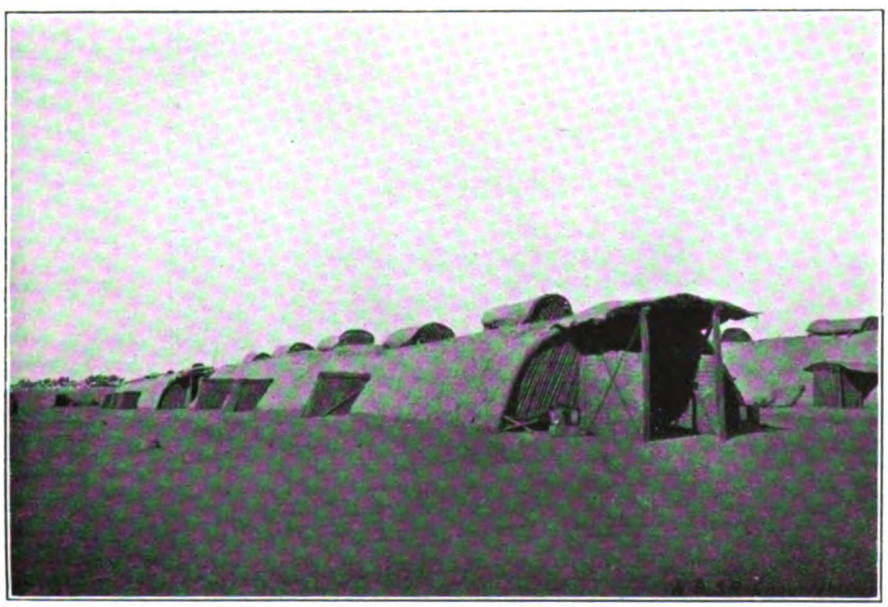


48.—Separate Hut Shelters for Cerebro-Spinal Fever contact cases, Carrier Hospital, Dodoma, East Africa.



49.—Exterior of type of Hospital Hut at the British Convalescent Depot, Amara. These huts of reed and chatai matting are hollowed out in the ground to a depth of four to five feet. Lateral openings provide light and air. They are comparatively cool in summer.

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50. Interior of type of Hospital Hut at the British Convalescent Depot, Amara. These huts of reed and chatai matting are hollowed out in the ground to a depth of four to five feet. Lateral openings provide light and air. They are comparatively cool in summer.

51.—Sandbag Bacteriological Laboratory at base of cliffs, Cape Helles.

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method for destroying fly eggs and larvæ in horse manure, and the remarkable mounds here shewn are the result of his efforts (*Fig. 45*). The heat of the manure is used as a lethal agent. Another device, which served to free a large hospital at Potchefstroom altogether of flies, was introduced by Lieutenant BABER, S.A.M.C., and has been described in the *Lancet* for March 30th, 1918. Here the larvæ migrating from the heap were trapped in gullies and destroyed, and any flies which happened to breed out found leafy branches soaked in arsenite solution conveniently placed for their refreshment and rest, the latter of the eternal variety.

BUILDINGS.

Lastly, a few words as to buildings. By far the most ingenious makeshift hospital ward I ever saw, was the fenestrated mud-brick erection constructed by Lieut.-Colonel OTWAY, R.A.M.C., of the 130th Combined Field Ambulance, on the road running north from Jaffa. I shew you external (*Fig. 46*) and internal views. Thanks to the use of mosquito mesh on the inner side of the fenestrations, and also over the doors and windows, this ward was absolutely free from flies and mosquitoes. It was partly hollowed out of the ground, but the portions left as bedsteads were of such a height that the patients lying on them could look out of the ward and see all that was passing. There was plenty of light and yet it was refreshingly cool. As I say, I never saw anything like it, and it deserves very honourable mention.

Some of the thatched bandas used as wards in the carrier hospitals in East Africa were truly works of art. Those in No. 1 Carrier Clearing Hospital at Iringa were really beautiful buildings and most comfortable, reflecting the greatest credit on Major ROBERTS, R.A.M.C., and his experienced architect, Staff-Sergeant EALES, S.A.M.C. I shew you the interior of one of them.

A trellised grape vine in a monastery close to the Jordan formed quite a good makeshift ward for the patients in an Indian Cavalry Field Ambulance (*Fig. 47*), while I would direct attention to the curious little shelters for cerebro-spinal contacts employed at Dodoma in East Africa (*Fig. 48*).

A method of warming a tent by a stove and chimney was in use on the Karun Front in Mesopotamia ; and, at Amara, the remarkable huts of the British Convalescent Depot dug out of the soil, roofed with reeds and provided with lateral ventilating shafts, were admired by all who saw them, and used by as many as could get into them (*Figs. 49 and 50*).

Finally, I may remark that no one who was ever inside the famous sand-bag bacteriological laboratory, tucked away out of shell-fire at the base of the cliffs at Cape Helles (*Fig. 51*) is likely to forget it, and that the remembrance of the huge packing case which had once held an aeroplane, doing duty at Dar-es-Salaam as an hospital laboratory, is not likely to fade.

I think I have said enough to prove that the army sanitarian has plenty of ingenuity, even if it is at times wrongly applied. Believe me, he has need of it, for he is often left in the lurch. Why is this the case? There are several reasons, but one is that the sanitarian has to go cap in hand to the Ordnance Department or the Royal Engineers and beg for material. Sometimes he will get it at once, sometimes after a long time, very often not at all. Let me tell you a true story which speaks for itself :—

A New Zealand medical unit on a certain island was badly in need of wood for sanitary purposes. Requisition after requisition failed. Finally, the Commanding Officer said to his Quartermaster, "I want you to indent on the Ordnance for a large supply of wood to make tombstones!"

"Tombstones!" said the Quartermaster, "what do we want with tombstones?"

"Never you mind," was the reply ; "do as I tell you."

Next day a cart, laden with wood of all shapes and sizes, made its appearance. There was plenty for the dead. There was none for the living.

Naturally enough the engineers, a very busy and capable corps, scarcely realise the paramount claims of sanitation. Like Martha, they are occupied with many things, and latrine construction does not usually appeal to them, nor do they always understand what is meant by efficient protection of a water supply. As I have said elsewhere, things will

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not be right until the Royal Army Medical Corps has affiliated to it a body of expert sanitary engineers, under its own control, to carry out the minor sanitary works which are so important. I know there are difficulties in realising such a scheme, but I believe they can be overcome.

In common with many of you, I have the honour to belong temporarily to the Royal Army Medical Corps, and I have seen again and again how the efforts of that corps are frustrated by its dependence on other departments. Too often it gets the blame when it is not blameworthy. Grant it a greater measure of autonomy and you will see what it can do. They say medical men are not, as a rule, good administrators. I deny that assertion. What are the qualities of a good administrator? Foresight, tact, discretion, firmness, energy and sympathy. What is prognosis but foresight? What is tact if a doctor does not learn it in dealing with his patients? Discretion is surely drummed into the medical man from his student days. He may not always be firm, but his experience at least teaches him to stiffen his back if he is to save life. Few men lead more energetic lives than the medical student, while sympathy is surely the hall-mark of our profession.

No, the doctor can administer if he only gets a fair chance.

At the same time I do not think that hygiene is yet priced at its proper value. I would advance a plea, an urgent plea, for its better recognition. Rightly or wrongly, decorations play a great part in army life. When a regular officer is introduced to a stranger in khaki, in nine cases out of ten his eye wanders first to the spot where what has been called the "herbaceous border" flourishes! He will never there see anything associated with sanitary work—I mean it has no distinctive reward. Comparatively rarely indeed has it any reward at all.

And yet the disciples of the goddess Hygeia—be they in the humblest or the highest positions—contribute greatly to the success of our arms, more especially in tropical countries. They have hard work, constant work, often dirty, dangerous and repellent work. Doubtless it is sufficient honour and reward to labour for the sake of the empire; but when so many distinctions are conferred, is it asking too much that the claims of the sanitarian should be considered, not, mark you, for his own sake, but to impress upon those ultimately responsible the fact that army hygiene is a very important section of army life?

Our friends the French are a wise and practical people—the French army has such a distinction. I understand it has been cheapened; but I maintain the principle is sound, and the recipients of any such honour should not be limited to the Medical Service, but should be chosen wherever an officer or man has shewn conspicuous zeal and energy in furthering sanitary measures.

There are many other questions of pay and rank, and, I may add, as far as sanitary sections are concerned, of mobility, but into these I may not enter.

I would only say that if ever the above suggestion—which I advance with considerable diffidence, and only because I am certain it is on right lines—if ever, I say, it bears fruit, then I hope the ribbon chosen for the decoration will bear the same colours as the flag of which I spoke—the white of purity, the blue of hope.

Discussion.

Colonel William Hunter: It has been my experience for two and a half years to be intimately acquainted and concerned with the conditions in the Balkan and other Eastern war areas—so well epitomised by Professor Simpson, and with the measures, sanitary and insanitary, adopted, so well summarised and illustrated by Colonel Balfour, with whom it has been my great privilege and pleasure to be associated in the work of the Advisory Committee for preventing epidemic diseases in the Eastern war areas.

During that experience nothing has impressed me more than the up-hill difficulties that had to be faced by the sanitary officers of the army, the number and variety of the problems of prevention they had to face with inadequate means, the number and variety of improvisations they had to create to enable them to carry on their work, and, lastly,

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subject to the limitation connected with the above difficulties, the remarkable degree of success which always attended their efforts *after these difficulties had been overcome*, both by concerted and individual initiative.

The magnitude of these difficulties was indicated in the figures given with regard to Gallipoli, viz., over 96,000 sick evacuated in the course of seven months in the autumn of 1915. The nature of these difficulties could, however, only be appreciated by those on the spot called on to deal with them. They were of a unique character, connected in the first instance with strategic considerations which necessitated the original landing, and the subsequent holding on to positions which gave no rest of mind or body, by day or by night; in which all landing of supplies had to be carried out in the hours of darkness on an open shore, without even landing piers; in which there was the greatest dearth of all material necessary for sanitary services; in which even the water supply—limited to a gallon or at most two gallons a day—had to be largely brought overseas from Alexandria or the Millwall Docks, London; and, lastly, in which all supplies were subject to loss by submarine attack: *e.g.*, at a critical moment a shipload of much needed material and appliances for sanitary work was thus lost.

To these circumstances were added those not only of utter fatigue of body, but plague of heat and flies and dust, which only those on the spot, as I myself have been, could fully realise.

The net result was the figure of sickness above quoted, made up chiefly of dysentery and diarrhœa (50 per cent.); "enterica" (typhoid and paratyphoid), (10 per cent.); supplemented later by "epidemic jaundice" (3 per cent.); reaching their maximum in September and October.

The figures were approximate, and served to indicate the magnitude of these infections that had to be dealt with in Gallipoli. They also indicated the degree of possible infection which was imported by the troops when evacuated to Egypt in January, 1916, and the problem of prevention which presented itself, both in that area and also in the Salonika area, during the following two years, where the work of the Advisory Committee lay. The result known to me was the following:—

Enterica.—During the year 1916, in Egypt, with twice, and, for a period, more than three times the number of troops, the incidence of enterica was checked from the very outset. It fell from its figure of 1,000–2,000 cases a month in Gallipoli (December, 1915) to about 400 a month in Egypt in the first quarter of 1916; about 250 a month in the second quarter, about 150 a month in the third quarter, about 70 a month in the fourth quarter, and, finally, to about 20 a month in the first quarter of 1917, at or near which very low figure it has since remained—a striking tribute to the value of inoculation.

Dysentery.—The fall here—uncontrolled by inoculation, as was the case with enterica—was also most striking, the total number of cases in the year 1916 being only about 6,000.

The general result was described by the War Office in official communications of July 7th and 11th, and August 11th, 1917, as follows:—

> *Egypt.*—"The health of the troops in Palestine is very satisfactory. Admissions to hospitals due to sickness for the first four months of 1917 shew a decrease in the annual ratio amounting to 145 per 1,000, as compared with the same period in 1916. The present ratio is lower than the average for Egypt during the five years preceding the war."
>
> *Salonika* (July 12th and August 11th, 1917).—"With regard to the health of the force, this year (1917) compares very favourably with the last. The health of the troops shews a marked improvement. In proportion to the strength of the force, admissions to hospital during July, 1917, shew a reduction to one-third compared with July, 1916; and deaths, from all diseases, a reduction of nearly two-thirds.
>
> "Dysentery and diarrhœa are appreciably less prevalent. The admission rate for fever, other than malaria, shews a reduction of nearly four-fifths.
>
> "The following table shews the number of cases of sickness per 1,000 men during the four weeks ending June 23rd, 1916 and 1917:—

	1916	1917
Dysentery and diarrhœa	20·55 ...	6·87
Enteric	0·37 ...	0·02"

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The above figures served to present the other side of the shield, viz., the results of preventive medicine work in the field, brought about, under difficulties, by the indefatigable labours of officers of the Sanitary Branch of the R.A.M.C. Although the status given to this sanitation branch of field work in the R.A.M.C. is probably higher than in most other armies, the experiences of the war had revealed in an exceptional manner the great, and, indeed, *primary* importance of this class of service. Its importance, I think, is not sufficiently indicated by the term "sanitary," applied to it, a term which in the general mind has come to be associated—all too exclusively—with the question of latrines and other measures of conservancy, and does not do full justice to the wide number and variety of problems, in all branches of science, which had to be faced in carrying out preventive medicine work in the field.

The title of "preventive" seems to me best to connote at once the character and the importance of that work—which more and more included not only knowledge of conservancy methods in the field, but a wide knowledge of branches of knowledge: entomology, bacteriology, protozoology, tropical diseases, etc., not covered by the term "Sanitary."

Various methods by which fuller recognition could be given to this branch of work had been touched on or suggested by the writers of the papers, and were worthy of fullest consideration.

Whatever organisation of this branch of the service might be adopted, an important precedent consideration in my judgment is to indicate by the title of the branch, viz., Preventive Medicine, the precise scope and importance of its work, coequal and coextensive with the great allied branch of hospital work concerned with the treatment of disease.

Numerous, and ingenious, and intensely interesting as were the inprovisations now necessary to carry on preventive work in the field, so well illustrated by Colonel BALFOUR in the demonstration he has given, it should be possible, in the light of our recent experiences, so to standardise our measures and *especially to arrange for their supply*, that dependence on hand-to-mouth methods need no longer be necessary.

The great sister branch of hospital work has not to improvise its supplies of beds, blankets, and material (tents, etc.), necessary for its work, but is supplied with these in abundance as required.

The needs of *Preventive Medicine* work in the field might well be and could be supplied by similar arrangements, which would give it full scope for its work, and not leave it dependent, as it now largely is on the supplies and mercies obtainable from so many other branches of the service—A.S.C., Ordnance, Engineers, as the case may be—upon whom so many and pressing demands come from all sources.

Professor W. J. R. SIMPSON: I congratulate Colonel BALFOUR on the graphic account he has given the Society of the sanitary and insanitary makeshifts he has encountered on his mission at the various centres of war. His experience is unique, and his recommendations for that reason will be most valuable to the Army. By his excellent and humorous description of the conditions which he saw, and which are well brought out by the photographs, he has managed to demonstrate, without offending any susceptibilities, some of the grave defects in the sanitary organisation of the armies in the East, and at the same time he has supported the views which I put forward at the last meeting.

Colonel HUNTER referred to the many difficulties, owing to the military situation, which preventive measures had to encounter. I am fully cognisant of these, but I am strongly of opinion that so far as the sanitary difficulties are concerned they need not have occurred, and that had an effective sanitary organisation existed, even in the case of Gallipoli—where it was found afterwards that no difficulty need have arisen in regard to a good supply of water and other sanitary considerations—there would not have been the terrible amount of sickness that prevailed.

Until the value of the work done, and which can be done, by the officers and men engaged in sanitary work, is more appreciated than it is even at the present time, there will be little chance that the organisation required to preserve the health of our armies

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will be given that importance and position which are needed for its efficiency. It must be acknowledged that a great advance has been made since the Boer war. The view, however, which Lord Wolseley held, and which found its way into the military Pocket Book, when sanitary officers were first appointed to the Army, that it was a sanitary fad, is still too prevalent among some people, and not infrequently acted on. Until this notion is thoroughly uprooted, and encouragement, promotion and honours are given to the Sanitary branch of the Service, in the same proportion as to other branches, there will be lacking the same enthusiasm for it. I have, however, great hopes that the experiences which Colonel BALFOUR, Colonel HUNTER and others have had of the defects of the preventive side of medical organisation in this war in the East, will result in great changes being effected on the lines I have indicated.

Then, in the next war—for I am not one of those who believe that war is going to disappear from the face of the globe—there will be a sanitary organisation ready, which will, as soon as war breaks out, send its Chief Health Officers and Staff, with material and powers of administration, to go forward and work in conjunction with the other military staff officers.

I am sure that in this way we shall have a much healthier army, and no repetition of the Gallipoli and Mesopotamian disasters

Lieut.-Col. A. CASTELLANI: I should like to congratulate Professor SIMPSON and Colonel BALFOUR on their very interesting papers.

In 1915-16 the Balkanic war zone was a hotbed of disease. In the early part of 1915 typhus was ravaging Serbia, one-tenth of the population was killed off by this scourge, one-third of the Serbian doctors died of it, and numerous victims occurred, too, among the medical men and nurses who responded to Serbia's call for help.

Relapsing fever was extremely common, and often was seen in patients recovering from typhus, this being due, of course, to the same insect carrying the two diseases.

Of malaria there were innumerable cases. I think I can say I saw more malaria in the Baltic zone in the two years I was there than in the sixteen years I was in tropical countries. The disease was extremely polymorphic, and could practically simulate any disease I know of. The treatment was difficult; it was doubly difficult there owing to the scarcity of quinine.

Typhoid, and especially paratyphoid, were quite common. I may, perhaps, be allowed to mention that the Serbian army adopted my combined vaccinations before any other army. Of Pappataci fever there were true pandemics. The dysenteries were extremely common. In fact the Balkanic zone was devastated by tropical diseases, and could be considered from a medical point of view to be a tropical country. Let us have a look now at the present condition of the same zone; nobody can deny that great progress has taken place. This progress is certainly due to the efforts of the medical departments of the British, French, Italian, and other allied armies, as well as the magnificent work done by men like Professor SIMPSON, Professor STRONG, Colonel HUNTER, and others.

Captain ANGUS MACDONALD: I thank Colonel BALFOUR for his most interesting paper, especially for its evident sympathy with the work of the practical sanitarian; and I ask the permission of the Chairman to put before the meeting a resolution in the name of Colonel Sir RONALD ROSS, who regretted his inability to be present: "That this Society forward direct to the Lord Chamberlain a copy of the paper of Colonel ANDREW BALFOUR, having marked the passages therein referring to a sanitary decoration."

Colonel S. P. JAMES: As one who has been much concerned with sanitary organisation in both peace and war in the tropics, I have read Professor SIMPSON's important paper with much interest. I am heartily in accord with his view that in tropical campaigns, even when we possess the knowledge which would enable us to prevent disease, the application of this knowledge usually fails from lack of an adequate sanitary organisation separate from the organisation primarily intended for curative needs. In this connection

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I hope it may not be out of place to make a few remarks upon the campaign in Mesopotamia, to which in January, 1916, I was deputed by the Secretary of State for India for the purpose of endeavouring to remedy certain grave defects on the sanitary side which came to light about that time. On this mission the late Dr. SANDWITH was to have accompanied me, but unfortunately for reasons of health he was unable to take up the appointment. I may preface my remarks by saying that in Field Service Regulations it is laid down that the first function of the personnel of the medical service is to preserve the health of the troops, and that in the Mesopotamia campaign this duty was especially important and urgent because (*a*) never before had so large an army operated under such unfavourable climatic and other natural conditions; (*b*) cholera, typhus, typhoid, relapsing fever, malaria and other epidemic diseases were endemic among the local inhabitants, and might at any time become so prevalent as almost to annul the fighting efficiency of the force; (*c*) many of the officers and most of the men were unacclimatised to tropical conditions, and had had no previous experience of campaigning. For these and other reasons it was to be expected that the necessity for a strong sanitary organisation would have been recognised in the beginning of the campaign. I found, however, on my arrival in February, 1916, that this was not the case, and that the urgency and importance of the sanitary and preventive branches of the medical services had been largely overlooked owing to *undue focussing of attention upon the purely curative branch* Sanitary sections had been depleted by detailing the personnel for duty in hospital; officers appointed for sanitary duties were being employed on routine office work; sanitary sections were in charge of very junior officials, who were without the necessary qualifications for this specialised class of work, and were without previous tropical experience; in general there was a complete failure to realise that for preventive as well as for curative work the staff must be proportionate to the work required.

In these circumstances the first task was to define the principles of the sanitary policy that should be followed, the second to create an independent organisation by which that policy could be carried out. In defining the sanitary policy we acted on the principle that the work required for preserving the health of the troops includes:

(*a*) Arrangements for obtaining intelligence regarding all influences affecting the health of the army.

(*b*) The preparation of a code of sanitary measures and rules based on the information supplied by these arrangements.

(*c*) Executive arrangements for carrying out the measures and rules.

(*d*) Arrangements for constant inspection to see that the measures and rules are everywhere carried out.

In creating the organisation for carrying out the policy, we laid stress on the view that sanitary administration and control can be efficient only in so far as it has expert inquiry and research for its starting point, basis and guide. Our line of action was, first, to detail expert sanitary officers and bacteriologists to investigate and report on actual conditions and needs at different parts of the line and in regard to different epidemic diseases; second, to formulate from the reports of those officers uniform measures to be adopted throughout the force, thereby to make such executive arrangements as would ensure the measures being carried out. I wish to emphasise that in providing the staff and means for carrying this line of action into effect, we had to neglect entirely sanctioned war establishments, and to provide an organisation entirely new to army medical regulations. Time does not permit me to give details of the organisation, so I will only say that the staff for the Central Administrative and Intelligence Branch, included a statistical officer, a sanitary engineer, two medical officers of health, a chemical analyst, an entomologist, and six special inquiry officers who were expert bacteriologists. Local intelligence and executive arrangements were also made without reference to sanctioned war establishments, and I believe the campaign was the first in which, in the tropics, an adequate sanitary organisation has been established quite separate and distinct from the organisation for purely medical needs. The arguments by which we were successful in obtaining an organisation for which there was no precedent, and which at first sight appeared unnecessarily large, were, first, that the conditions under which the force was operating

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were altogether unexampled; second, that there were enormous possibilities of disasters if disease among the troops got beyond control; third, that the sanitary requirements asked for were relatively very small in comparison with those always considered essential for the curative side of the medical service.

Captain J. GRAHAM FORBES: I am in complete agreement with Professor SIMPSON'S criticism of the conditions under which the sanitary officers are called upon to carry out their important duties in connection with camp sanitation and water supply. And particularly in countries such as the Balkan area, where they are faced with the difficult problems of organising and supervising preventive measures against malaria and dysentery in dealing with the mosquito and fly pests, I would emphasise the vital necessity of sanitary officers being provided with proper authority and military rank. After two years' experience in Macedonia I recognise only too well the difficulties of efficient sanitary work without these very necessary powers.

The hospital unit in which it fell to my lot to undertake the duties of sanitary officer was the first of several sent out to be attached to the Serbian army. On arrival at Salonika we were apparently quite unexpected, and the object of our coming seemed to partake of the nature of a surprise. No arrangements had been made as to our destination, and there was consequently some delay in settling on the choice of a site. Decision was eventually arrived at as the result of a day's trip up the line, some fifty miles in the direction of Monastir, by the officers in command of two units, under the guidance of the French authorities, and the only possible camping ground out of several inspected was selected at the end of what must have been a particularly trying and hot day. The choice of a site was necessarily dependent on strategic conditions, but, as events proved, suffered from a hasty and imperfect acquaintance with the particular locality. The chief advantages attached to it were the close proximity of the main railway and road to Monastir towards the west of the allied line of defence.

Arriving as we did in midsummer, it was most important that immediate steps should have been taken to attempt to carry out some scheme of anti-malarial measures and drainage, but owing to insufficient supply of labour and other causes it was impossible to put in hand any serious or co-ordinated effort to remove or reduce the danger of malaria, which was to take heavy toll of hospital patients and personnel.

It was not until the late spring of 1917 that the Mission Antipaludique Française, together with the augmented working parties of the sanitary sections of the two units, was able to take the problem in hand of draining the surrounding areas and clearing the dense growth. These efforts bore fruit in the summer and autumn of 1917 and 1918 in a considerably diminished incidence of fresh cases of malaria, and this diminution was undoubtedly assisted by the freer use of mosquito netting in the men's lines, the issue of which in 1916, and until late in 1917, having been quite inadequate.

In addition to their anti-malarial measures in the terrain, the French mission carried out investigations into the incidence of malaria among the natives of the surrounding villages, and ascertained the spleen index among children to vary from 40 to 70 per cent. of all those examined. Quinine was also administered to all those whose blood showed the presence of parasites.

Water Supply.—A better acquaintance with the locality at the time the site was originally chosen would have ensured a far more satisfactory supply than we were able to depend on. For at the foot of the hill-slopes, some three miles across the tongue of plain from the selected site, was a copious spring of good water of constant volume issuing from the limestone rock and flowing in a series of pools to join a main river. The spot, moreover, was free from swamp and risk of flood, and therefore provided less breeding ground for the mosquito. The cost of communication with rail and main road would have been inconsiderable compared with what had to be incurred in furnishing a means of supply from the small source on which the camps in the chosen site were dependent. This source was a small spring which came to the surface some 300 yards from the hospital boundary and followed the course of an old Turkish aqueduct for a short distance before discharging into a small basin two feet in diameter, and was thence carried off into one of the many little streams.

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Sanitation.—The sanitary officer was, unfortunately, not ordered to accompany the party sent in advance of the unit to prepare the camp site. His duties, therefore, commenced under difficulties, with a very insufficient labour staff.

Being dependent on the French authorities and situated at a considerable distance from Salonika, there was much delay in the provision of materials to construct latrine sheds. In the meantime, and for a period of three months, we were obliged to rely solely on the latrine trench system, which required unceasing watchfulness to insure the free use of earth covering.

After the lapse of three months the permanent latrine sheds, with concrete flooring, were complete, and provided with portable drums or buckets with lids.

Experience soon taught that the simpler the system for the Serb the better the chance of an approach to sanitary cleanliness. The plank seat and back-rest, fitted in well-meaning spirit by the French engineer, proved to be not only superfluous, but put to wrong use, which led to much fouling. Both were, therefore, eventually removed, and the Serb was able to adopt the attitude he was most familiar with, free from the hindrances of a supporting seat.

For the disposal of excreta it was not found practicable to incinerate, and the simpler plan was adopted of removal in the Crowley Cart for burial in large pits about a mile from the camp, where free use was made of cresol and chloride of lime each day to disinfect the contents, surface, and sides of the pits. Excreta from the dysentery and enteric wards and latrines used by convalescents from those wards, in addition to being treated with cresol, were boiled in a large vat in the annexe to the special latrine, and the boiled residue was removed by the cart for burial with the rest of the camp excreta. Large boxes with compartments for sanitary bins, containing cresol solution, were provided in the annexe for disinfection of bed-pans and infected linen, and a special boiler was also used for further disinfection of pans and linen. The linen was then removed to the Thresh disinfector for steam sterilising.

Solid camp refuse was collected in sanitary bins and petrol drums and disposed of at first partly by burial in pits—a very unsatisfactory system—and partly by incineration; but as soon as it was possible to build several incinerators of closed pattern all solid refuse was burnt.

It was at first thought possible to dispose of liquid refuse by means of soak pits, but owing to the lack of absorption by the clay soil and the high level of subsoil water the soak pits rapidly filled up, and for this reason they also proved quite unsuitable for urinals. In the case of urinals it was found best to provide petrol drums well cresolised, and to remove the urine by the sewage carts for burial with the rest of the excreta in the pits a mile from the camp.

The liquid waste from kitchens and bathing tents was at a later date collected in a special soil cart for emptying into one of the streams at a distance from the camp—a tributary of the river which eventually emptied itself in the marsh several miles to the east. The separation of liquid from solid refuse was a constant source of trouble, for both British and Serbian orderlies alike were very slow to realise the impossibility of burning the wet sodden mass that was frequently brought to the incinerator. Similarly the liquid soil cart, and in early days the soak pits, were frequently choked with solid refuse, in spite of notices and instructions to the contrary, and the provision of separate bins labelled respectively "solid" and "liquid" refuse in both English and Serbian

Flies and the Camp Surroundings.—The fly problem in the East is one of, if not *the* most difficult and harassing of tasks which beset the thorny path of the sanitary officer. The conditions surrounding the camps during the summer and autumn months after our arrival in July, 1916, offered every attraction to the winged pest invading our lines, and taxed the energy and vigilance of our sanitary section to the utmost. Both in kitchens and latrines the daily use of cresol solution by means of spray and broom proved of great value in reducing and keeping away the flies. In the wards and mess tents a spray solution containing 1 per cent. formalin, carbolic and eucalyptus was found to be fairly effective as also the use of fly papers and wire fly traps. Unfortunately owing to the regulations in force, arsenite of soda was not available, and sodium salicylate and formalin

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solutions placed in open dishes proved of practically no use. In the immediate vicinity were small camps occupied by some of the allied units and by Macedonian labourers where the standard of sanitation left much to be desired, and necessitated frequent visits for the removal and abatement of nuisances responsible for the attraction and propagation of the fly pest. Likewise the encampment in the neighbourhood of troops of almost every nationality and of passing natives greatly increased the chances of fly prevalence and infection. During the subsequent summer and autumn, 1917 and 1918, anti-fly measures were rigidly enforced, and the marked diminution in the numbers of flies was generously attributed to these efforts, but was more probably largely due to climatic and seasonal influence of a prolonged drought.

Col. W. G. KING: A perusal of the papers by Professor W. J. SIMPSON and Lieut.-Col. A. BALFOUR leads to the conviction that whilst on the Western Front with but little movement of the troops, and with their bases practically in England, sanitary efforts have been eminently successful, this cannot be said to be the case in certain of the subtropical and tropical areas in which military operations were conducted. The conditions which existed, in spite of the loyal efforts of the officers concerned, could not be satisfactorily met by the sanitary service of the Army as organised.

Professor SIMPSON suggests desirable and reasonable administrative reforms; Colonel BALFOUR deals with the logical outcome of such reforms in rendering sanitary efforts no longer dependent on the concurrence, or forethought, of the Ordnance or Engineer Staffs. Both hold that while there should be close intercommunication between the curative and sanitary branches of the Army Medical Service, each should be self-contained.

The administrative recommendations of Professor SIMPSON are compatible with the practical opinion of Colonel BALFOUR, that an essential part of a scheme of reform shall be that the Sanitary Branch of the Service shall have under its command a technically trained body of men, capable of relieving the Engineer Corps fully of duties connected with minor sanitary works – as contrasted, I presume, with major sanitary works which would fall to that Corps, in consultation with the Sanitary Service.

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Obituary.

Dr. R. P. COCKIN.

We regret to announce the death on December 9th, 1918, of Dr. Reginald Percy Cockin, a fellow of the Society from 1908.

Dr. Cockin was born at Hull in 1879, and studied at Cambridge, taking his M.A. degree in 1906 and his M.D. in 1913. His medical studies in London were carried out at the London Hospital. After graduating in medicine he acted as casualty house-surgeon of Hull Royal Infirmary, and after that entered the West African Medical Service. He was appointed district surgeon in Southern Nigeria, and later on medical officer of the Niger Cross expedition in 1908-1909. In 1910 he was transferred to Cyprus, acting here as a district medical officer and examiner under the Pharmacy Act. In 1913 he was posted to Grenada, West Indies, where he held the posts of resident surgeon to the Colonial and Yaws hospitals and also did bacteriological work. After this he joined the Staff of the London School of Tropical Medicine. In 1915 he was appointed a temporary Lieutenant in the R.A.M.C., and went to Egypt with Lieut.-Col. Leiper and Captain J. G. Thomson to study bilhariasis. After his promotion to Captain, owing to ill health, he was obliged to resign his commission, and resumed his duties at the London School of Tropical Medicine. He was appointed Assistant Medical Entomologist, and assisted Colonel Alcock in his duties as Deputy Director of the School in the absence of Lieut.-Col. Newham, the Director, on service in East Africa.

When the special venereal clinics were formed in London by the Local Government Board, Dr. Cockin initiated and started the one at the Albert Docks Hospital, and acted as its Director. He quickly built up a large practice and made it a great success.

Dr. Cockin attended the meetings of the Society regularly, and took a lively interest in the papers read before it. Of a retiring nature, he did not speak much during the discussions, although his wide tropical experience gave him every right to do so. He was a keen and hard worker, and his knowledge of tropical medicine was well exemplified in the excellent reviews he used to contribute to the *Tropical Diseases Bulletin*. As one of the younger men in Tropical Medicine, he had a future before him, and his loss will be severely felt by his colleagues at the London School of Tropical Medicine as well as by his numerous friends elsewhere.

The following is a list of his published works, that on the treatment of yaws by salvarsan being a very important contribution :—

"A Further Report on Work Done in Connection with Ankylostomiasis at the Colony Hospital, St. George's, Grenada (Windward Islands)." Report to the Secretary of State for the Colonies, July, 1912.

"Report on the Treatment of Twenty-two cases of Yaws, by Salvarsan Injections, at the Yaws Hospital, St. George's, Grenada, W.I." *Jl. Trop. Med. and Hyg.* 1912, Sept. 16. Vol. XV., No. 18, pp. 277-278.

"Ankylostomiasis in Grenada," *Parasitology*, 1913, Apl. Vol. VI., No. 1, pp. 57-67.

"Treatment of Yaws by Intramuscular Injections of Salvarsan." A Report on a Series of 45 cases treated at the Yaws Hospital, St. George's, Grenada, B.W.I." *Lancet*, 1913. Dec. 6, pp. 1609, 1610.

"A Case of Rat-Bite Fever Treated Successfully by Injections of Novarsenobillon," *B.M.J.*, 1918. Feb. 16, pp. 203, 204, with Dr. G. C. Low.

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TRANSACTIONS

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Proceedings of a Meeting of the Society held on Friday, January 17th, 1919, at 5.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Surgeon-General Sir DAVID BRUCE, K.C.B., F.R.S. (*President*), in the Chair.

MALARIA CONTRACTED IN ENGLAND.

BY LIEUT.-COL. S. P. JAMES, I.M.S. (ret.).

The principal observations on malaria contracted in England, which were made during 1917, were published last June in a collection of reports to the Local Government Board. It will be remembered that the total number of locally contracted cases reported among all classes of the population in that year was 178, of which 19 were in the Navy, 136 in the Army, and 23 in the civil community. The experience of malaria contracted in England in 1918 will be similarly published by the Local Government Board in due course, but I am permitted to make a general reference to its known extent and distribution in the civil population, to be supplemented by the contributions which are promised to the Society in regard to naval and military data.

The following statement gives the number of civilian cases of malaria which are at present known in this country, with the localities in which they were detected. The total

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is 67, of which it is believed that 43 were contracted in 1917, or earlier, and 24 in 1918:—

SUMMARY OF KNOWN CIVILIAN CASES.

County.	Place.	Contracted in 1917 or earlier.	Contracted in 1918.	Totals.
Suffolk	Southwold	1	0	1
Essex	Tilbury	3	0	3
,, ...	Walton-on-Nase	2	1	3
,, ...	Dovercourt	1	1	2
Kent	Queenborough	27	5	32
,, ...	Sheerness	0	2	2
,, ...	West Minster	0	4	4
,, ...	Sheppey (Rural)	8	3	11
,, ...	Isle of Grain	0	6	6
,, ...	Sandwich	1	1	2
Salop	Shrewsbury	0	1	1
		43	24	67

It will be seen that most of the cases occurred in north-east Kent, near the mouth of the Thames, an area comprising the Isle of Grain and the Isle of Sheppey—which includes the rural district of that name,—the town of Sheerness, the small borough of Queenborough and the hamlet of West Minster. In regard to the cases in these localities and in Essex I give the following information:—

Queenborough town.—In Queenborough town, at the beginning of 1918, there were eleven residents who had been notified as having contracted malaria during 1917. By visits to the town and the appointment of Dr. SEELLY as Special Malaria Officer on the civil side, these cases were kept under continuous observation, and in April and May, when several of them suffered from relapses, they were provided with medical attendance and treatment and with mosquito curtains free of cost. When these arrangements became known, several other persons, whose cases had not previously come to notice, reported themselves to Dr. SEELLY, so that by the end of May the number of known cases had risen to eighteen. Systematic enquiry for cases and the taking of blood films were then begun, with the result that, by the end of the year, the total number of malaria patients known in the town amounted to thirty-two, of whom it is believed all except five had their first attack in 1917 or previously. Of the new cases, one was discovered on the 23rd July, the remaining four in October.

Sheerness town.—In 1917 no case of malaria was reported from the town, but in 1918 there were two, both being believed to be new infections. Both were discovered early in September.

West Minster.—This is a small village consisting of two rows of houses alongside the railway about midway between Queenborough and Sheerness. In 1917 no new case was reported from the village, but in 1918 there were four, of which one occurred in August, two in September and one in October.

Sheppey Rural District.—In 1917, in the area called Sheerness East, which adjoins Holm Place Hutment Military Camp, eight cases were found. Three new cases were reported in 1918—one in July, one in August and one in September.

The Isle of Grain.—During the week ending October 5th, 1918, two cases of malaria were detected by Surgeon HULL, R.N., among civilians in the Hoo Rural District. The patients were two children, aged 1½ and 7 years, in a cottage a few hundred yards from the headquarters of the R.A.F. Station in the Isle of Grain. At a visit of enquiry on the 15th October, three other cases came to notice, and Surgeon HULL reported a sixth case on November 1st.

Sandwich.—There was one civilian case in 1917, and one civilian case has been detected in 1918.

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Essex.—Enquiries made by Lieut.-Col. WILKINSON in Essex have brought to notice seven cases of locally contracted malaria, namely, three at Tilbury, two at Dovercourt, in the Harwich Urban District, and two at Walton-on-the-Naze. Apparently five of the seven cases were contracted in the autumn of 1917, but they were not discovered until they relapsed in 1918.

I may now refer to the following subjects of epidemiological and clinical interest in connection with the cases in the north-east Kent area.

The 1917 epidemic in Sheppey.

In 1917, nearly all the locally contracted civilian cases which were discovered in England occurred in the Queenborough and Sheerness area of the Isle of Sheppey as part of a well-marked epidemic which affected both troops and civilians in that locality. According to our present information, the total number of civilian cases in that outbreak was 35, of which eight occurred in close association with the military cases at the Holm Place Hutment Camp, and 27 occurred in Queenborough town. The epidemic prevalence of malaria in the locality was first detected among soldiers in the Holm Place Camp, and the origin of the new military cases there was attributed to the presence in the camp of many malaria infected soldiers who had returned from Salonika. The new infections among the troops were with the Salonika strain of the benign tertian parasite, and the occurrence was an outbreak, not of indigenous malaria, but of exotic malaria, due to importation of the Salonika parasite. It seemed clear also that the civilians who lived within or in the immediate vicinity of the camp were also infected with the Salonika strain of the parasite, for their illnesses were of the same severe type as those of the military cases, and the incidence of the disease among them, as regards time and place, could be explained only on that view. We concluded, therefore, that in the Holm Place area, as regards both troops and civilians, we had to do solely with a newly imported exotic infection, not with an infection which could be correctly styled indigenous.

But when the investigation was extended to Queenborough town we found cases which could not be ascribed with probability to an origin from military foci, and although in these cases, as in those near Holm Place, the parasite was of the benign tertian variety, the clinical symptoms and signs were much milder. Moreover, the detailed enquiries which were made in all these civilian cases enabled us to refer some of them to an origin from other civilian cases in their immediate neighbourhood, and in a few instances to civilian cases which had originated before troops began to return from Salonika. On these findings, and with the local knowledge that as late as 1865–70, "nearly everyone in Queenborough suffered from ague," it appeared probable that in this instance the infections were with the indigenous strain of the parasite which had never entirely disappeared from the town, and that the condition in 1917 was an epidemic rise of prevalence in a locality where true indigenous disease was already endemic.

I think therefore that in the Sheppey outbreak of 1917, there were two independent foci, one (originating at Holm Place) being a focus of exotic malaria, the other (originating in Queenborough town) being a focus of true indigenous malaria.

During the last three months of 1918, we had an opportunity of observing in the Isle of Grain a similar but smaller outbreak of true indigenous malaria, which in this instance occurred in the absence of any malaria infection among neighbouring troops or naval garrison. I refer to the six cases which I have already mentioned as having been detected in the small village of Grain. The outbreak was among four families living in a row of dilapidated cottages near the Royal Air Force Station. The occupants of one of the cottages numbered six, of whom three (it was said) had suffered from ague in previous years. One of them was an old lady, who gave a quite accurate account of both tertian and quartan ague, and described the relapses from which she used to suffer. She said that most of the local inhabitants had had ague at one time or another, and that as a rule they did not call in a doctor, but treated themselves with quinine powders or pills

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bought from a chemist. Among the personnel of the Air Force stationed in the locality there was no old case of malaria and no new case had occurred.

As a contrast to this example, I may mention a small outbreak of locally contracted malaria which occurred in 1917 among troops in Aldershot area. This was clearly an example due exclusively to the importation of the exotic disease, and it was confined to cases of infection with the exotic strain of the parasite. It did not spread to civilians, and so far as we have been able to ascertain the indigenous disease does not occur in the area.

From these observations, I think it may be inferred that as regards malaria in England, we have to deal with two different, and as a rule, independent conditions: the one arising from the importation of exotic malaria and due to a foreign strain of the parasite, the other being true indigenous malaria, due to a parasite which has never entirely disappeared from this country.

Clinical differences between the exotic and the indigenous disease in England.

It is unfortunate that we are not as yet able by blood examination to ascertain whether a patient is infected with the exotic or with the indigenous race of the benign tertian parasite, but clinically there is, as a rule, a considerable difference between the two classes of case. The civilians who were infected at Holm Place Camp (undoubtedly with the Salonika parasite) were seriously ill and suffered many relapses. But civilians infected in Queenborough town (presumably with the indigenous parasite) suffered only very mild attacks, and relapses have been rare. Indeed many of these cases would have escaped discovery if house-to-house enquiry and the routine taking of blood films had not been the rule. A number of examples indicating the mildness of type of the indigenous disease came to our notice. Mrs. ——, of Castle Street, Queenborough, was discovered during a routine examination of all the occupants of the house. She said that "occasionally she had cold shivers, but did not take much notice of them." She had not at any time ceased to carry on her usual work. Parasites were present in her blood. Agnes C——, aged 13, of Harold Street, Queenborough, answered the door when we visited the house and was engaged in helping her mother with house-work. She appeared bright and cheerful, but her mother said she was paler than usual, and that she intended to send her to Guildford for a change. We took a blood specimen and found a considerable number of parasites in it. The parents of one of the affected children at Grain were treating him with small doses of ammoniated tincture of quinine, because they happened to have it in the house, and it did not seem worth while buying quinine powders as the case was mild.

These experiences are very different from those of civilian patients who were infected with the exotic (Salonika) strain of the benign tertian parasite. It suffices to mention that in at least three of the civilian infections contracted at Holm Place Camp, the patients were so alarmed at the severity of their illness, that they left the district as soon as they were sufficiently recovered to do so.

At present I must be content to record, without attempting to explain, the observation that true indigenous malaria in England is now a milder disease than formerly, and a milder disease than that is exotic malaria contracted in England. The practical importance of the observation is, first, from the point of view of patients, that it is certainly preferable to be infected with the indigenous parasite rather than with the exotic type; second, that for ascertaining the incidence of the indigenous disease and for the purpose of a campaign against it, it is not sufficient to rely upon a system of notification based on clinical symptoms—especially when those symptoms do not alarm people to the degree that makes them willing to go to the expense of calling in a doctor. Our experience in Sheppey shews that for the discovery and prevention of cases of the indigenous disease, the first step should be the provision of free medical attendance and quinine; the second, the institution of special enquiries in every area in which the occurrence of the disease may be suspected.

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Indigenous Malaria a Household Disease.

I now refer to some observations in connection with the spread of the indigenous disease in Queenborough town. The cases were not scattered in a random manner throughout the town, but were definitely localised to certain streets and houses. Most of them were in three groups, namely: (1) the Harold Street and Stanley Avenue group, (2) the Comyn Terrace group, and (3) the Castle Street and Woodhall Terrace group. In Harold Street there were cases in the houses numbered 19, 27, and 29; in Comyn Terrace in the houses numbered 1, 4, and 6; in Woodhall Terrace in those numbered 2, 5, and 12; and in Castle Street in those numbered 20, 24, 25, 26, 27, and 32. The localised character of the distribution is more clearly indicated by the incidence of cases in particular houses; and on this subject the findings provide some striking examples of "malarious houses" quite comparable to the well-known instances of "yellow fever houses," which were such a puzzle to epidemiologists before yellow fever was known to be carried by the domestic mosquito, *Stegomyia fasciata*. In No. —, Castle Street, there have been five cases at intervals during 1917 and 1918; in another house there have been three cases, and in four houses there have been two in each. In the first of these houses the occurrence of cases at intervals suggested the need for a blood examination of all the occupants. This was done on July 18th, 1918, and the landlady, Mrs. ——, whose case has already been mentioned, was found to be an apparently healthy malaria carrier. Unknowingly she was probably the original source of infection to several lodgers in her house.

The strictly localised character of the distribution of cases in the town, and particularly the incidence in houses, indicates that the species of mosquito concerned has the habit of remaining most of its life within or in the immediate vicinity of the house where it obtained its first meals of blood. It will be very fortunate if further knowledge of the habits of this species enables us to classify it among the strictly household or domestic mosquitoes against which effective measures are practicable. I hope that Mr. GROVE will supply further notes of the habits of this mosquito in this respect.

Relapses and Anopheline Conditions.

On the subject of relapses in cases of indigenous malaria, I have to note that among 20 cases, continuously observed since their original attack in 1917, 6 had no relapse during 1918, and that among the remaining 14 cases there were 19 relapses, of which 3 occurred in April, 7 in May, 4 in July, 4 in August and 1 in September. If we can continue to obtain detailed records of relapses among all new infections, the results may be of some assistance in an attempt to forecast the next epidemic year. To the same end Mr. GROVE has obtained numerical records of the anopheline conditions in each month of the year in different localities. It is important that in Queenborough in 1918 careful search failed to reveal the presence of any adult anopheles in the houses during April and May, when most relapses occurred. During the first week of June a few male and female adults of the first brood were found in stables, but the number increased slowly, and it was not until July 18th that adults were first found in dwelling-houses in the town. One conjectures that in Queenborough during 1918 there would have been more new cases of malaria if the emergence of the first brood of adult mosquitoes had coincided with the spring rise in relapses.

All the observations I have narrated relate solely to infections through the agency of *maculipennis*, which is the only species concerned with the spread of malaria in Queenborough. The role of *bifurcatus* and *plumbeus* in the spread of malaria in England has still to be worked out, and upon it I have no observations to record this evening.

Perhaps other members can help us with definite observations on this subject.

Measures.

On the civil side, the measures taken in 1918 to deal with the disease were based on an endeavour to discover and control cases and carriers as early as possible. For this

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purpose compulsory notification of cases has been supplemented since May by making personal enquiries and taking blood films in households and at school inspections. On the discovery of a case or carrier, steps are taken to ensure :—

(*a*) That the patient during the attack shall rest in bed five days, taking 30 grains of quinine a day.

(*b*) That thereafter he shall take 30 grains of quinine once every ten days for three months.

(*c*) That he is supplied with a proper mosquito curtain, is taught how to use it, and regularly sleeps under it.

(*d*) That the patient and other occupants of the house make a practice of killing all adult mosquitoes which they find in the bedrooms and other rooms of the house.

Patients are visited daily during the first five days and thereafter every tenth day. Surprise visits are also made to ascertain if the curtains are being correctly used. Blood films of all discovered cases are taken at regular intervals.

Discussion.

Mr. A. J. Grove, M.Sc.: Colonel James has asked me to mention some of the results which have been obtained from some investigations which I have been making during the past year for the Local Government Board, upon the prevalence of anopheline mosquitoes. It will be remembered that in the early part of 1918 some information regarding anophelines in England was published in a series of reports submitted by the Local Government Board, and at about the same time the Trustees of the British Museum issued a map shewing the recorded distribution of anophelines in England and Wales up to that time. Useful as this information undoubtedly is, it serves to emphasise how little is known of the real prevalence of anophelines, and more particularly our ignorance regarding the habits of the insects under natural conditions in this country; and it was in the hope of throwing some light upon these two subjects that the investigations were commenced.

Three areas were selected (1) Great Wakering in Essex, (2) New Romney in Kent, and (3) Berrington, near Shrewsbury in Shropshire. The first two had each a previous history of malaria and the third had not, and were selected to serve as a sort of control. Each area was visited in rotation every three weeks, and similar investigations were carried out in each.

The first set of observations with which I will deal is an attempt to estimate numerically the prevalence of the adult anophelines in these three areas. Previous experience has shewn that, during the daytime, adult anophelines, particularly *Anopheles maculipennis*, are to be found hiding in cowsheds, stables, etc.; in fact such buildings seem to form their natural diurnal resorts. A number of cowsheds, etc., was selected in each area, and at each visit counts were made in each of them on two days. The method adopted was, after a preliminary search to locate the insects, to record the number which could be counted in half a minute. At the same time a sample of the insects was taken to determine the species present. Curiously enough, although *A. bifurcatus* has been recorded several times from such buildings, yet throughout these observations the only species of anopheline met with in the stables was *A. maculipennis*.

The results obtained were not quite similar, but in general the numbers found gradually decreased until the end of May was reached, but at the beginning of June a sharp increase occurred, which marked the appearance of the new brood. From this point the numbers fluctuated until a maximum was reached about September. (This applies more particularly to the Great Wakering and New Romney areas, while in the case of Berrington the maximum was reached in June, though there was a distinct rise in numbers during September). From the end of September the numbers began to decline.

With regard to *A. bifurcatus* and *A. plumbeus*, numerical observations on these lines

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could not be carried out owing to the fact that neither of these insects resort during the daytime to places in which such counts could be made. It may be stated, however, that *A. bifurcatus* has been found, sometimes in numbers, in all of the areas, and *A plumbeus* at Great Wakering and a locality about three miles from Berrington.

The next set of observations with which I will deal refers to the prevalence of the larvæ. In each of the areas a number of breeding-places was selected, and these were examined at each visit and records kept of the conditions found It was found impracticable to devise a method by which numerical counts could be made, such as those which had been given for the adults of *A. maculipennis*, but a general estimate was made of the prevalence of the larvæ on each occasion. *A. maculipennis* larvæ were found from the middle of May until the beginning of September. *A. bifurcatus* larvæ were found in decreasing numbers until the end of April, after which they apparently disappeared until the beginning of June, from which time they have been found continuously up to date. *A. plumbeus* larvæ were not found at all, and it is evident that this species does not frequent the type of breeding-place favoured by *A. maculipennis* and *A. bifurcatus*.

From Colonel JAMES's paper it is clear that one of the most important questions upon which knowledge is necessary in connection with malaria in this country, is that of the factors which are concerned with the invasion of buildings, and particularly houses, by anopheline mosquitoes. In this connection it will be useful to refer once more to the results obtained from the periodic counts in the cowsheds, etc. An examination of the individual records shewed that some of the buildings were evidently more favoured by the *A. maculipennis* than others, and from a critical examination of the character of the building, particularly with reference to the character of the roof and the lighting, it has been possible to deduce factors which seem to determine whether a building is likely to be favoured or not. These factors in brief are: (1) the continued use of the buildings by animals; (2) the absence of draughts, particularly near the roof; (3) comparative stability of temperature; (4) the intensity of light.

I will now give some examples of the occurrence of anophelines in houses. It was hoped that some numerical records, similar to those obtained for cowsheds, etc., could be obtained for houses in the areas, but, owing to the reluctance of the British householder to admit a stranger into the bedrooms and other private portions of his house, time after time this was found to be impracticable However, at each visit to the areas I endeavoured to stay at a house within the area, and in these cases some interesting results were obtained.

At Great Wakering the house at which I stayed, during the visits from April to August, was situated in the centre of the fields of a market-garden, and mosquitoes of various kinds were to be found swarming in the fields around the house on suitable evenings, and *A. bifurcatus* was particularly numerous at times. Yet on no occasion was any anopheline found in the house until the last day of the visit, about the middle of August, when, as I was sitting at breakfast, about half-past eight, a female of *A. plumbeus* attempted to bite my left hand, which was resting on the table.

Another case of interest occurred at New Romney. The house at which I stayed there was a detached one, surrounded by a garden, and the latter adjoined the yard of the Station Hotel, in which was a large stable where *A. maculipennis* abounded. The bedroom I occupied overlooked the garden on the hotel yard side, and the window was invariably left open, but in spite of the close proximity of numbers of *A. maculipennis* none were observed in the bedroom during the visits made in April, May and the beginning of June. However, on the 25th June I was awakened at 5 a.m by the buzzing of a mosquito, which eluded capture at the time, but in the morning a female *A. maculipennis* containing blood was found on the wall. At the next visit, about the middle of July, quite a series of invasions took place. On the morning of the 16th July, a female *A. maculipennis* was found in the room, and there was a mark on my forehead which was possibly a bite. The next morning four males and seven females were found in the bedroom. Irritation of the head and face had been noticed in the early morning, but no bites were visible when I got up. The insects were left undisturbed, and by the morning of the 18th only four females remained, the others having left during the previous evening and night. A mark of a bite was noticed on the left thumb.

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A further case of interest was an attempt by two females of *A. maculipennis* to bite me whilst I was changing in my bedroom at the Station Hotel, New Romney, at 4.30 p.m. on the 9th October.

Colonel JAMES has dealt at some length with the cases of malaria at Queenborough, and on the 24th September I examined, through the kindness of Dr. SEELEY, a number of the houses in which cases of malaria had occurred. *A. maculipennis* was found in many of the houses, especially on the ceiling of the landings.

What, then, are the factors which control the invasion of houses by *A. maculipennis*? My own view—arrived at rather from general considerations, and probably subject to modification as our knowledge of the subject increases—is that the invasion of houses is associated with the swarming, for pairing purposes, of the insect. The operation of pairing completed, the immediate object of the females is to seek a meal of blood and the males a hiding-place for the coming day. It is known that *A. maculipennis* swarms usually at a height of twenty feet or so from the ground, and if the swarming-place happens to be close to the open window of a bedroom containing a human being, it is natural for both males and females to enter it in pursuit of their various objects. Whether they will stay there for any length of time depends upon whether the room or the house is suitable or not, and now the factors which have been enumerated above in connection with the observations on the cowsheds come into operation. This appears to me to offer a feasible explanation of the findings at Queenborough. The houses examined were nearly all built on a particular plan. The entrance hall gave place to a staircase which led up to a central landing, from which the bedrooms at either end and along one side opened. The ceiling of this landing supplied three of the four factors, viz., absence of draught, stability of temperature, and, when the bedroom doors were closed, diminished lighting, in fact, almost complete darkness. The people occupying the house would serve as a substitute for the first factor. This may explain why the insects were found in such a large percentage of cases, and, combined with the records from the cowsheds, certainly supports Colonel JAMES's view that the insect does not stray far from the place where it obtained its first meal of blood except for the purpose of laying its eggs.

Hitherto all the instances of biting given have concerned *A. maculipennis* and *A. plumbeus*, and in order to complete the series I will give an instance of *A. bifurcatus* biting. On August 14th I went, together with Mr. R. F. BURTON, into a pine wood on his estate at Longner Hall, Salop. Almost as soon as we had settled ourselves in a spot near a small pool of stagnant water we were attacked by numbers of various species of Ochlerotatus. After a while their attacks subsided, and then isolated females of *A. bifurcatus* were observed approaching, with a curious zigzag flight, evidently intent upon a meal. During the time that we were there, 5.30 to 7 p.m. (summer time), four females were caught after they had attempted to bite us. Mr. BURTON informs me that he has had similar experiences with *A. bifurcatus* at different times, varying from eleven in the morning till evening. This observation is interesting because it throws light on the conditions under which *A. bifurcatus* may attack human beings, although it does not commonly frequent houses.

Other matters and details will, of course, appear in a report to the Local Government Board, but I have selected these as bearing on some of the points mentioned by Colonel JAMES.

Surgeon-Captain P. W. BASSETT-SMITH: In 1917 there were nineteen cases of malaria contracted in England reported from Naval bases. These were summarised in the Local Government Board's Report on Malaria (No. 119).

During 1918 I have had nine fresh cases reported in men who have never been abroad, or if abroad (two cases), never had previous attacks of fever—one left Australia in 1913 and the second was in the Mediterranean in 1916.

One case, in a boy, was contracted at Shotley, in Suffolk, near Harwich. It is stated that mosquitoes were at the time abundant in the low-lying marshes, and specimens of *A. maculipennis* were sent to me. The fever was of an intermittent type. He was the only one affected, and on enquiry no cases of malaria nearer than Southwold (twenty miles

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distant) were traced. The second case was an ordinary seaman from Burntwick Island, Sheerness. The man had been in the Mediterranean in 1916, but had never been ill. He was employed on shore at the time and mosquitoes were troublesome. Five of the other cases were from the Isle of Grain, Kent, which returned eight cases last year. Marshland and stagnant pools, intersected with dykes, surround the station, and mosquitoes are generally abundant, and many living specimens were sent to me of *Culex pipiens* and *Anopheles maculipennis*. All but one case occurred in the early part of the year, April, May and June.

There were two cases from Eastchurch, Kent, both in September. This station returned six cases last year. It is surrounded by marshland and ditches, and in the hot weather swarms with mosquitoes.

There are six other cases which could be said to have definitely contracted the disease at the Isle of Grain in 1917, and relapsed or were reinfected during the year. All the cases were proved to be malaria by finding the parasite in the blood, which was always *P. vivax*.

The Naval Medical Officer of the Isle of Grain station states that undoubtedly cases of malaria have occurred in several of the villages around, and that splenic enlargement is an early and marked symptom of the disease. He quotes an instance of a family in which three were attacked by the fever, and he found in each an enlarged spleen. The cases tend to be very localised, those working in the shed of the Engineering Construction Camp being most affected, and in this area anopheline mosquitoes were frequently found. It is also noted that they were particularly abundant in the stables of a certain farm. He came to the conclusion that there was an endemic pre-war form of malaria in the district, as special precautions were taken to prevent the spread of the overseas type of infection, and an enquiry in September, 1918, shewed that there were only five old malarial cases on the station. These dated back to 1915, and none had suffered from recent attacks of fever, though, with a constantly changing personnel, infection might have been present from time to time, but none of the new cases were traced directly to any of these known forms. Cases which came under my treatment, however (sent on to the Seamen's Hospital, Greenwich), shewed frequent relapses and much anæmia, as is common in the Salonika infected men.

The prophylactic measures taken were: every case as soon as diagnosed was given quinine, put under a mosquito net, and drafted as soon as possible to hospital or to a non-malarial station. Any rating known to have a history of recent malaria is immediately sent out of the station.

Every endeavour was made to destroy the larvæ by paraffining the pools, etc., around the living quarters, and the edges of these were kept free from long grass, but, the area being so large, it was impossible to do more than this.

With regard to the cases which were contracted on the station in 1917, and either relapsed or were reinfected in 1918, the time of onset in all was the early part of the year. In most of these cases the previous attacks were not diagnosed or reported at the time.

TABLE.

	Cases.	Onset. April.	May.	June.	July.	Aug.	Sept.
Shotley ...	1	—	—	—	—	—	1
Sheerness ...	1	—	—	—	—	1	—
Eastchurch ...	2	—	—	—	—	—	2
Isle of Grain	5	1	2	1	—	—	1
	9	1	2	1	—	1	4
Relapsed or Reinfected Cases.							
Isle of Grain	6	1	3	2	—	—	—
Sheerness ...	1	1	—	—	—	—	—
	7	2	3	2	—	—	—

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Captain ANGUS MACDONALD, R.A.M.C. (T.): To achieve coherent realisation of malaria indigenous in England requires appreciation of three main factors of infection—the infective carrier, the soldier returned with malaria from endemic country; the transmitting vehicle, the anopheline mosquito in England; and the victim, soldier or civilian, who has not been out of this country or has not been infected with malaria in any country.

Confining consideration to malaria, imported and indigenous, amongst troops in England, I present briefly the facts of importation, transmission and indigenous occurrence recorded, in their connection, during the period 1916 to 1918.

With the factors of infection the conception of prevention is inevitably linked up, and brief reference will be made to the modifying influence of preventive measures on indigenous incidence.

Therapeutics, coming as it were after the event, I leave alone.

I. Malaria Carriers.—With the return, in the winter of 1916-17, of Expeditionary Force men from Eastern seats of war, came the anticipated recognition of relapsing malaria: and in April, 1917, the special control instituted by the War Office definitely, by the issue of Provisional Instructions for Treatment, directed the care of sufferers from malaria.

These instructions had in view the wise and efficient treatment of men and their refitting, as well as the safety of other troops and civilian populations. Already in January, 1917, centres in the Commands had been selected for special treatment of malaria cases; but it had not been considered administratively justified that the fact alone of having suffered from malaria, or of being probably liable to relapse, should prevent men discharged from hospital from returning in natural course to their units. From the start, therefore, malaria carriers were distributed in the Commands throughout the country.

The further expectation of the occurrence in England of indigenous malaria was realised in the autumn of 1917, and with the guidance of these occurrences it was determined by the War Office, in consultation with the Local Government Board, to schedule as "dangerous areas" those districts in which cases had arisen, embraced chiefly in Romney Marsh, in Sheppey and the Isle of Grain, and in Sandwich. For reasons of prevention, special provisions, in addition to those already in operation, were agreed on for control in these dangerous areas, to be applied to other districts if occasion should arise by extension of the occurrence of indigenous malaria.

In July, 1918, it was considered advisable to remove malaria carriers from dangerous areas, and in August it was further decided to concentrate all malaria carriers in the Commands at home for transference to France, where camp conditions were available more readily than in England for aggregation and localised control. It is probable that before these removals were effectively consummated, any breaches in the preventive armour had been taken full advantage of by *Anopheles maculipennis*.

The distribution in the months May to September, 1918, throughout the dangerous areas of actual known malaria carriers was:—

1918.	Sheppey and Isle of Grain.	Romney and Lydd.	Sandwich.
May	491	86	20
June	552	91	32
July	550	24	57
August	349	23	25
September	46	33	38

These figures are minimal, and the amounts are subject to individual interpretation of the designation "carrier," which arbitrarily implied all who had relapse within sixty days. Different camps carried varying strengths of returned malaria personnel who might be liable to relapse, and of different ranks amongst whom relapse if slight was not revealed. For instance, it has been notorious that amongst officers it was customary to "carry on" in spite of malaria relapse, which was usually self-treated; and at Lydd especially officers under training were in large number. On the grounds suggested, therefore, it is justly

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assumed that the efficiency of the personal control of carriers was various and in general partial.

II. Anopheline Transmitter.—During 1917 and 1918 considerable addition has been made to the knowledge of the distribution in area and in numbers, and the habits of the Anophelinæ throughout England, Scotland, Ireland and Wales. Much is yet to learn.

Precise detail of observations is elsewhere recorded. Here and now it may be permitted briefly to summarise observations, mainly personal, and inferences tentatively arrived at for purposes of preventive administration, so far merely as they bear on the matter of infection.

1. *A. maculipennis*, *A. bifurcatus*, and *A. plumbeus* have been found—*maculipennis* with great frequency and in greatest numbers; *bifurcatus* less frequently and fewer; and *plumbeus* rarely.

2. *Maculipennis*, adult, is found the whole year round; it may be called the domestic mosquito, its winter dwelling being in the warmth and shade of occupied stable buildings and its warm weather wanderings bringing it in contact with man in the home.

3. *Bifurcatus* is a creature of the open, rarely coming into human dwellings.

4. *Plumbeus* is sylvan, breeding in tree holes.

5. Probably the usual food of the female of all three is the blood of animals; *maculipennis* has been taken frequently feeding on cattle in stables.

6. All have been recorded biting man.

7. *Maculipennis* (adults and larvæ or potential breeding places) has been found in immediate association with each case of indigenous malaria recorded. *Bifurcatus* and *plumbeus* have not been found in such association.

8. *Maculipennis* alone is found in any number in human dwellings, and from its habits is accepted as the most probable usual transmitter of malaria in England.

9. Comparative observation shews no difference of distribution and incidence of *A. maculipennis* in the years 1917 and 1818; and meterological conditions differed but in slight degree.

III. Indigenous Malaria—Occurrence in 1917.—The cases of indigenous malaria recorded amongst troops in England during 1917 number 163. Obviously this is a minimal record.

An individual investigation of these occurrences in 1917 was not undertaken personally by specialised officers; detailed criticism of indigenous cases is confined to those arising during 1918 when individual investigation was undertaken.

Occurrence in 1918.—A record is presented of 61 cases of indigenous malaria occurring amongst troops in England in the year 1918, containing brief detail in tabulated form of the points essential to the determination of the infection and of indigenous origin:—1, Serial Number; 2, Regt. No.; 3, Rank; 4, Name; 5, Unit; 6, Location at date of onset; 7, Probable location of infection; 8, Residential history; 9, Malaria history; 10, Evidence of infection; 11, Infection factors (*a*) carriers, (*b*) mosquito, (*c*) larvæ and breeding-places. With the exception of No. 8 the cases have all been personally investigated by the recorder, the others by other responsible officers.

Some consideration is offered of these indigenous cases and of the factors of endemicity in England.

Evidence of malaria has been established by expert pathologists.

Evidence of indigenous origin has been accepted or established by the recorder after exhaustive epidemiological criticism of all factors known to be concerned.

The *Infective foci* have been assumed to number 29 a minimum.

The incidence and distribution in the years 1917 and 1918 are shewn in Table I.

The *Seasonal Distribution of Indigenous Occurrence and Infection* is presented in Table II.

In Table III. evidence is to be found bearing on facts and assumptions relating to indigenous occurrence in 1918.

There is apparently undeniable evidence of infection in early May. From the recorded temperatures, and from the estimated temperatures, over thirteen 16-day periods

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from 19th April, anopheline plasmodial development under natural conditions would have to take place at temperatures ranging from 44° F. to 51° F.

Adventitious incubation within heated military huts is postulated and was probable in all four cases. In June and July the few cases occurring may have had similar origin It may be noted that May and August infections are definitely recognised in temperate countries. June and July supply few cases. Assuming in a temperate country the necessity for incubation at indoor temperature of heated rooms to produce May infections, it is evident that social custom would explain the rarity of infection in the June and July months, in which ordinarily fires will have been left off in dwellings. By August, normally, anopheline plasmodial development finds ample temperature for occurrence.

The possibility of *accidental infection* by transfusion of blood of a malaria carrier has in all cases been eliminated.

Endemicity of Malaria in England. Definitions applied to malaria :—

1. *Indigenous* implies simply origin in the country.

2. *Endemic* means belonging to the country, and the use of the term demands that all factors causative are always and can be naturally present.

3. *Autochthonous* - frequently used by French writers — implies even a closer association than aboriginal with the country. The term is quite inapplicable to malaria, but might, for all we know, apply to *A. maculipennis*.

4. *Epidemic* is wide incidence in a point of time of infectious disease.

From evidence accumulated it is suggested here that the factor definitely determining the occurrence of malaria in England is the importation of carriers ; that at the present day there is no evidence to support the belief that malaria is endemic (as defined, or continuously endemic) in England ; that the evidence accepted to demonstrate that malaria at one time was endemic in England, may equally be taken to prove that it depended for its reappearance on the importation of carriers ; and that while excessive importation of carriers may from time to time give rise to an outbreak of indigenous malaria, the absence from continuous operation of one of the infective factors will prevent the establishment of malaria endemic in England.

The last previous recognition of malaria in epidemic form was in the years 1857 to 1859. In 1856 and later years there is evidence of large importation of ague especially from Eastern Europe. The coincident abnormal elevation of temperature in the summers of 1857–1858–1859 enhanced infectivity, and recognised epidemics occurred.

Malaria seems quickly to have disappeared after 1860, when summer temperature receded.

Spontaneous and therapeutic recovery from malaria takes place in a definite, but not established, percentage of cases infected in a period short of eleven months. Eleven months—August–September to July–August—are in general required for the vital continuity of malaria infection under normal meteorological conditions in England. Under these normal conditions there is evidence that the carrier volume brought forward from year to year is insufficient to maintain malaria endemic in England. Hence the immunity of the past sixty years, over which we can cast the seeing eye.

The *importation of carriers* determines the *fact* of indigenous malaria ; *protracted elevation of temperature* coinciding determines the incidence. Extensive epidemic will result from an optimum of coincidence.

IV. Preventive Measures—Quinine Administration.—As a prophylactic, quinine has not been used or recommended officially in England.

Screening. - Huts have been screened for the segregation of malaria carriers on all camps in recognised dangerous areas Screening has varied in satisfactoriness. There is no record of *maculipennis* being found within screened huts at Sandwich. At Sheppey, *maculipennis*, male and female, have from time to time been taken in small numbers in screened huts.

It has been stated above that evidence suggests that, for various administrative reasons, it may be accepted that in all camps some carriers escaped detection and segregation within screened huts.

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Pathological Work.—Expert means of ready diagnosis were provided in all areas concerned, and prompt recognition of case and carrier distinctly aided prevention.

Entomological Research.—To enhance the value of field observations, which have been systematically pursued by various officers, an experimental entomological laboratory has been carrying on work, of which the results will appear in due course.

Anti-mosquito Measures.—These consisted of direct attack on the adult *A. maculipennis* in its winter haunts and throughout the year in all haunts, and on the breeding-places of *maculipennis* and *bifurcatus*.

For administrative reasons it was found possible to deal with the Sandwich area with a directness and completeness not readily applicable to other areas. *The results obtained at Sandwich are claimed to issue from the more intensive sanitation there carried out*—See Table I.

The local records of mosquito presence, the general expression of diminution of nuisance, and, most of all, the morbidity record in face of increased infectivity, demonstrate again that deliberate and sustained attack on the transmitter is the way to deal with malaria.

Give the Sanitarian a chance, and the Therapeutist will find malaria removed from his consideration.

Summary Conclusions.—1. There is evidence that malaria has occurred in the British Isles in past times.

2. There is evidence of the occasional local extension of malaria to epidemic magnitude.

3. There is evidence pointing to the absence of continued endemicity of malaria from the British Isles.

4. All factors essential to the continued endemicity of malaria are not continually present in the British Isles.

5. The brevity of the annual infective period, together with the protracted period between each annual infection time, within which spontaneous and therapeutic recovery may take place, prevent continued endemicity.

6. Malaria, indigenous, but locally discontinuous, may be maintained year by year by the sporadic importation of "carriers" under normal conditions of movements of population.

7. Under conditions of extensive importation of carriers wide infection may suffice to produce epidemic, which, owing to causes specified in statement 5 above, will subside with rapid progression in seasons.

8. Epidemic occurrence and continuance depend upon a coincidence of factors, the optimum of coincidence being—

(*a*) Large importation of carriers to anopheline areas;
(*b*) Ample incidence of anophelines;
(*c*) Abnormally high temperature and its abnormal continuance;
(*d*) Association of "carriers" with susceptible populations.

9. In any defence against malaria in the presence of extensive importation of "carriers," sanitary measures directed to the limitation of the anopheline are of proved paramount importance.

TABLE I.

COMPARISON OF INCIDENCE AND DISTRIBUTION IN 1917 AND 1918.

Situation.	1917. Number of Cases.	1918. Number of Cases.
Sheppey	68	31
Isle of Grain	3	3
Sandwich	69	6
Lydd ...	1	2
Aldershot	4	4
Elsewhere	18	15
	163	61

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TABLE II.

Seasonal Distribution of Malaria Occurrence and Infection.

Occurrence.		Infection.	
Date, 1918.	No. of Cases.	Probable Date	No. of Cases.
May ...	3 ...	May ...	4
June ...	2 ...	June ...	2
July ...	1 ...	July ...	3
August ...	22 ...	August ...	47
September..	29 ...	September..	4
October ...	4 ...	October ...	1
	61		61

TABLE III.

	Station A, East Coast.				Station B, Midlands.		
1918.	Max.	Min.	Mean.		Max.	Min.	Mean.
May ...	60·9	47·2	54·0	...	63·7	43·9	53·8
June ...	63·9	48·4	56·0	...	64·9	44·6	54·7
July ...	66·8	53·7	60·2	...	67·7	50·7	59·2
August ...	68·0	55·1	61·5	...	69·3	51·9	60·6

Mr. A. Bacot: I would suggest, with regard to temperature tables in connection with malaria in this country, the importance of recording the *maximum*, together with some indication of the *period* during which the day temperature is high enough to allow of the development of Sporozoits within the mosquito.

The mean temperature may convey an erroneous impression in clear sunny weather, when hot days are followed by cold nights. Such conditions, while greatly lowering the mean, would probably only delay and not prevent development of the malarial parasite, as would be the case in cloudy weather, when cool days are followed by a relatively small night fall of temperature.

In connection with Captain Macdonald's communication, the question occurred to me as to whether there is a possibility of the feeding instincts of *A. maculipennis* having altered within historic time in accordance with the extensive change that must apparently have taken place in the sources of its food supply. Prior to (say) the seventeenth century, the hibernating females must have been much more restricted to human blood than is now the case, when stalled horses and cattle are frequently more numerous in proximity to their haunts than human habitations. It would be interesting to test by experiment whether mosquitoes, hibernating in stables or cowsheds, are as eager to attack man as those living in human habitations.

Sir Ronald Ross: I do not suppose that Colonel James considers that the hypothesis that there are two strains of benign tertian parasites now in England is to be taken as proved; but he has been quite right to make this very interesting suggestion, and I hope that further investigations on the matter will be made. For example, I would suggest that Captain John Thomson, R.A.M.C., who has been recently doing much cultivation work on the malaria parasites, should be asked to make cultures of the two suggested strains, in order to see whether there is any morphological difference. I might suggest, also, that volunteers may be found who would be willing to be inoculated!

The second point in Colonel James's paper concerned the "spring rise" in malaria. This is of course an old story. The Italians observed it many years ago, and hence called benign tertian by the name of "spring tertian." They shewed that there is always an increase of such cases in March and April, long before the anophelines begin to bite. Subsequently I examined very carefully the Indian statistics, and found a similar rise occurring almost everywhere in the months of February and March, also before the Indian anophelines begin to bite. I made a special study of the conditions in the

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Punjab in India, where this phenomenon is marked, and I discussed the matter in my book on "The Prevention of Malaria." Recently Captain ANGUS MACDONALD has given, I believe for the first time, what appears to be the true explanation, namely, that at this season the mosquitoes, which are now ready to come into active service at the beginning of the spring, are encouraged to do so by the heating of the houses. It was previously thought that this spring rise was due to the hibernating mosquitoes remaining infected during the whole winter, but recent observations in Salonika suggest that this is not at all true.

I regret that owing to the lateness of the hour I cannot say more about Colonel JAMES's interesting paper.

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MALARIA, WITH REFERENCE TO

(1) THE DANGER OF IMPORTED ANOPHELINE INSECTS,

(2) AN UNUSUAL BREEDING GROUND.

BY

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I.

This article deals with the malarial problem in Khartoum, a town which is situated at the junction of the Blue and White Niles, and which has a hot and dry climate with a very small rainfall.

The Southern Sudan is highly malarious, while the Northern Sudan, until recent years, was not nearly so much infected; but the steadily increasing number of pump irrigated farms and the very high Nile of 1917 converted it into a very hot-bed of malaria in that year.

The natural sources of infection for Khartoum are—(1) River pools, (2) garden and household collections of water, (3) wells, (4) rain-water pools, (5) irrigated farms.

The rain, though very small in total aggregate, comes in a few heavy storms, which often flood streams and produce many pools which may last weeks.

Besides the five sources mentioned, there is a large traffic in steamers, native boats, and trains coming from highly malarious districts.

Before preventive measures were adopted there is abundant evidence that Khartoum was intensely malarious, but since steps, first inaugurated by Lieut.-Colonel ANDREW BALFOUR, C.M.G., while M.O.H., have been taken to deal with the problem, the town has been kept remarkably free from malaria, and a mosquito is rarely seen or heard.

The total population of Khartoum and Khartoum North is about 33,000, and the recorded malarial cases have been taken as follows:—

Oct. 1st to Sept. 30th.	Khartoum.	Khartoum N.	Total.	Approximate Incid'ce per 1000.
1911–1912	56	19	75	2·27
1912–1913	31	16	47	1·40
1913–1914	51	17	68	2·06
1914–1915	58	16	74	2·24
1915–1916	5	2	7	0·21
1916–1917	109	14	123	3·75
1917–1918	264	36	300	9·09

The records from 1911–1916 are highly satisfactory, while the increases for 1916 to 1918 were produced by—(1) the high Niles, especially that of September, 1917; (2) the great increase in native craft bringing in wood, due to the shortage and exorbitant price of coal.

The number of native boats now bringing wood into Khartoum is about forty-five to every one prior to the war.

These boats nearly always come from highly malarious districts, and journey slowly down or up the river to Khartoum, often tying up to the banks during the night.

Thus anophelines come on board to feed off the crew, and, when fully gorged, seek

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the nearest shelter, which in most cases is the cargo, especially wood cargoes, where they remain undisturbed until unloading occurs.

The anti-malarial measures adopted are briefly given :—

(1) The towns are divided into areas, to which a man of the mosquito brigade is appointed.

He inspects weekly every house, garden, well, etc., in his district.

(2) Every adjacent farm is inspected weekly.

(3) All the river banks and pools are inspected weekly.

(4) Mosquito clearance stations have been formed on the Blue Nile, east and west of Khartoum, and every river craft has to call for inspection, the clearance of water and live mosquitoes, when possible by SO_2 gas, or cresyl, and an attempt is made to bolt mosquitoes from exposed cargoes by smoke producing machines.

(5) The prosecution or imposition of a fine in every instance when mosquito larvæ more than three or four days old are found in domestic utensils, etc., or in places where ordinary care would have prevented their occurrence.

The fine imposed usually corresponds to about 10s.

(6) Constant check inspections are made by the British Staff to see that the work is thoroughly done.

Except under such circumstances as a very high Nile or very extensive rains, Khartoum can be, and is, kept practically free from malaria, and, were it not for the imported infective insect, the records would be much more satisfactory, and it is to call further attention to the importauce of such sources of infection, in places where active anti-malarial work is done, and where the success is to some degree nullified by the advent of such insects, that this article is written.

The more one studies the malarial problem the more one is forced to recognise that in combating the source of the disease the battle must not only be fought locally but must be extended to regions far beyond what has been, and often still is, regarded as sufficient, and when traffic from highly infected regions exists the imported imago has to be reckoned with.

The following facts are recorded dealing with imported mosquitoes : - -

(1) The area of Khartoum and Khartoum North adjacent to the river is far less densely populated than that further away, and yet the former has supplied a far greater number of malarial cases than the latter.

For instance, between October 1st, 1916, and January 20th, 1917, there were ninety cases, believed to have been locally contracted, of which fifty-five occurred within 150 yards of the river bank amongst 2,900 inhabitants, while of these there is evidence that thirteen or more occurred from a flight of mosquitoes from a wood boat, and that of the remaining twenty-two it appears highly probable that six were contracted through infective mosquitoes brought in by trains. We have demonstrated on several occasions the presence of mosquitoes in incoming trains.

During the period under consideration the wind is blowing from the northern direction, thus causing Khartoum to be more affected than Khartoum North.

The sources of infection might be neglected breeding places in pools, gardens, houses or steamers and boats ; but for ten months in the year mosquitoes are practically never seen, yet all these sources are present all the year round, except that the steamers and boats, though running all the year round, are more numerous with the high Nile, and are found only to harbour anophelines to any degree during the period when breeding places exist in large numbers outside the controlled area.

(2) During the period just mentioned river craft are often found, when passing through the clearance stations, to harbour considerable numbers of anophelines.

They are very rarely found to be breeding on board, though culex and stegomyia frequently do so.

The anophelines are nearly always females, and undoubtedly come on board for human blood, and not to breed.

Except in holds and cabins, which can be closed and fumigated, the problem of how to deal with them efficiently, and at a reasonable cost, is very difficult : at present we try to bolt them with smoke outside the town.

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The map shews that cases tend to collect round unloading stations, which are marked X.

(3) Many times it has been found that a few mosquitoes appear in a house near the river bank, which has been free from them for months, and this has almost invariably coincided with the advent and tying up of a steamer or native boat opposite to the house, while no local neglected breeding places could be found on most thorough examination.

For instance, on March 14th, 1918, two very large barges, ladened with wood, arrived opposite the Burri Water Works, having come from the White Nile, which was at that period teeming with mosquitoes.

Prior to this, for some considerable time very few mosquitoes had been noticed at the waterworks, but immediately after unloading was commenced mosquitoes appeared in considerable numbers.

The Chief Engineer's servants' quarters are immediately opposite the unloading place, and in their bedroom we found—

On March 16th, 18 female anophelines;
" " 17th, 7 " "
" " 18th, 5 " "

and after this date only one every few days, and within ten days all had disappeared.

In the adjacent houses very few could be found.

We have found wire mosquito flappers the best method of killing off such mosquitoes, and during the winter of 1917, and early in 1918, two men were employed daily to visit all servants' rooms and the latrines along the front; by this means large numbers were killed off.

Curiously, in the latrines, quite ninety per cent. were male culices, the female culex being seldom found, and the anopheline but rarely.

This observation has been often corroborated locally, and the love of the male culex for the latrine bucket appears to strongly indicate that the male culex loves feeding off human excreta if he gets the opportunity.

(4) The mosquitoes known to breed locally are *Pyretophorus costalis*, *Culex fatigans* and *Stegomyia fasciata*.

Species, which do not breed locally, have been found in houses near the river banks and in incoming trains:—

(a) Tæniorhynchus.

(b) Cellia pharœnsis.

(c) A very large, dark coloured culex, unidentified at present, but known to be a marsh breeder.

(5) One very instructive example of malaria caused by anophelines from a wood boat is now recorded. *(Vide* Plan I.)

On December 22nd, 1916, malaria began to appear amongst the soldiers of the Military Works Department and the Sirdar's escort.

Ten cases appeared between December 22nd and January 2nd, and then the outbreak abruptly stopped.

This area is inspected weekly, and a very careful check inspection failed to shew any neglected breeding place.

About the same time two small epidemics occurred amongst the British troops and the Egyptian battalion at Said Pasha Barracks.

Ten British soldiers developed the disease between December 19th and January 3rd, and then the disease claimed no more victims, while eight cases occurred at Said Pasha Barracks between December 22nd and December 30th, and then no more cases appeared.

Careful inspection revealed no missed breeding grounds, and Said Pasha Barracks lies surrounded by desert.

No pools were found to have larvæ more than two or three days old, and the farm on the other side of the river was found to be free.

It appeared possible that these three little outbreaks were connected, and investigation elicited the following facts:—

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On December 7th a nuggar, loaded with wood, having evaded the Mosquito Clearance Station, arrived opposite the Military Works Department, and was unloaded by the soldiers between 2 p.m. and 6 p.m. on that day.

The wood had come from a highly malarious place on the Blue Nile.

Now the only bodies of men living near this unloading station are:—(1) The Military Works Department; (2) Sirdar's escort: and both were infected.

The civil public works department buildings are immediately opposite the unloading station, but are unoccupied at night, except for two watchmen, one of whom went down with malaria.

Pools, owing to the falling Nile, had commenced to form late in November opposite the Burri Waterworks, a little E.N.E. of the British barracks, but were kept under supervision.

On December 9th thirteen pools were found to be heavily infested with anopheline larvæ two to three days old.

On following up the history of this wood boat we found it had tied up on the evening of December 6th opposite to these pools, and had remained there until the morning to pass through the bridge.

At this date the wind was blowing from the N.E., and there can be no doubt some mosquitoes flew on shore, some laying eggs in these pools and causing the cases of malaria, namely, two at the Burri Waterworks, ten at the British barracks, all occurring in the N.E. portion, and that some flew on to Said Pasha Barracks across the desert, some 1,000 yards distant, but only infected two rooms (*Vide* Plan I.), these, except for one, being the nearest to the British barracks.

It will be noted that the first occupied room, *A*, was unaffected, though 95–100 men sleep here, and the reason was because the N.E. windows, facing direct to the wind, were kept closed, while the windows of *B*, being sheltered from the direct effects of the wind by the projecting room *A*, were left open.

The windows of room *C*, facing N.E., were also kept closed, but there is a large arched opening between rooms *B* and *C*, allowing mosquitoes to pass freely from one to the other.

Room *B* developed five cases, four being near the most north-easterly window, while room *C* only developed three cases.

Rooms *D* and *E* were not affected, proving the well-known tendency of the mosquito to seek the nearest occupied dwelling.

In all these epidemics the malignant tertian parasite was present.

It seems probable that farther on in the town five other cases were caused by stray mosquitoes from this source, as they cropped up singly at this period and no local breedings out could be discovered; and, further, if due to a local breed out, the cases would have been more numerous and grouped together.

Six cases of malaria occurred in the railway employees' quarters during these four months, and these were almost certainly due to infective anophelines brought in trains.

II.

"*An unusual and, it is believed hitherto unrecorded, type of breeding place of the anopheline mosquito* (probably *Pyretophorus costalis*)."

The inundation around Khartoum, caused by the Nile flood of 1917, was very extensive, and totalled many square miles.

Tuti Island was largely flooded, and would have been almost completely submerged but for ramparts thrown up to keep the flood from the pump cultivated area.

The south-easterly portion of this island consists of large sand dunes with extensive intervening hollows. (*Vide* Plan II.)

The Nile reached 16·83 on September 22nd, and again on September 27th, from which date a very rapid fall took place for some weeks, but by the end of November the river level was being maintained fairly stationary.

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The rapid fall drained off the large lakes that had been formed between the sand dunes and many of the pools, while we were able to deal with those remaining so as to render them harmless.

However, the amount of water sucked up and contained by the sand dunes must have been very considerable.

As the river fell large sand and mud banks appeared, but, except in one place, they all rapidly hardened on the surface, leaving scattered pools here and there.

At this one spot a large mud flat formed, some 150 yards long by some 30 yards broad.

Now the silt brought down by the Blue Nile in flood is very fine, and remains in suspension in the water for a very long time, and thus produces such a fertile plain as that of Egypt.

This mud flat was formed by this fine silt, and was of the consistency of thick pea soup.

It was not connected with any pool, there was no visible water, though the surface glistened in the sun.

On entering it one's feet went through without appreciable resistance until one was above one's knees in mud.

On taking up the fluid in a vessel it remained as fluid, and for a very long time.

Plan II. shews a rough section and plan of this flat.

The place was not examined for mosquito breeding, as it was concluded it was not a suitable breeding place; but on November 24th, 1917, one of the mosquito brigade men, while examining the pools on the island, tried to take a short cut by wading through this mud flat.

As he floundered through his dipper happened to enter the mud, and by force of habit he looked at the contents, and was surprised to find an anopheline larva five to six days old.

He carefully cleared the dipper and took other specimens, and found several more larvæ of different ages.

Mr. MURRAY, the Chief Sanitary Inspector, after confirming their presence, reported it to me, as the condition seemed a very difficult one to deal with.

I visited the place, and anopheline larvæ of various ages were found in considerable numbers, and this was confirmed later on, in the presence of Major R. G. ARCHIBALD, D.S.O., of the Wellcome Tropical Research Laboratories, and Mr. H. KING, the Government Entomologist.

On November 29th anopheline pupæ were found in fair numbers.

The question arose how one was to deal with this breeding ground, as both larvacide and oil tended gradually to spread on the surface and then slowly moved down towards the river, as the mud flat very gradually shelved in this direction. The perpetual and continuous spraying of oil or larvacide would soon have run into a very large sum at war prices; besides our stock would not have allowed of such a procedure.

Consequently I had a trench dug round the whole periphery, just outside of the mud flat, so as to try and intercept the water flow, and in doing this we came across the secret of the mud flat.

After digging through some ten inches of earth we came to a sand stratum, and some two feet below this a rocky stratum, which passed out from the island towards the river and is apparently restricted to this site.

In the deeper portion of the sand water gushed out like a spring the whole length of the canal adjacent to the island, and was undoubtedly fed and maintained by the vast amount of water still being given up by the sand dunes and island.

Elsewhere, there being no rocky stratum, the water took a deeper level, enabling the sand or mud to consolidate on the top and form banks

It appears probable that the rocky stratum has a slight slope from the periphery to the centre, thus forming a shallow basin and allowing the water to escape but slowly to the river.

Major E. E. AUSTEN, of the British Museum, kindly wrote to me about this, and from his letter I understand such a breeding site has not before been described.

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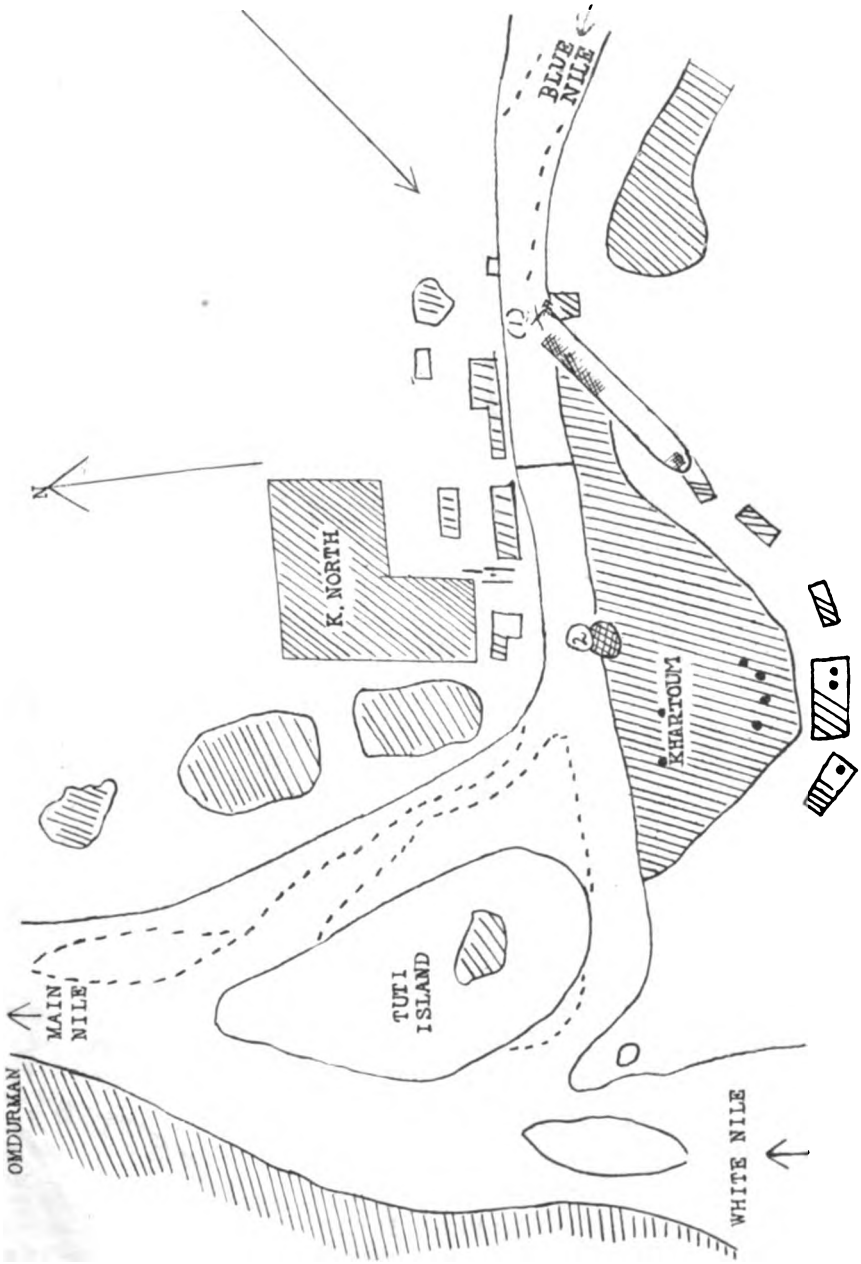
After digging the trench and intercepting the waterflow, and leading it off direct to the river, the flat fairly rapidly consolidated and dried up.

I regret we cannot definitely say whether anophelines bred out from this site, but if they could pupate, it appears probable that the adult insect could emerge from the pupal case. No attempts were made to breed out insects from larvæ or pupæ taken, but *Pyretophorus costalis* is the only anopheline which has been found to breed locally.



PLAN I.

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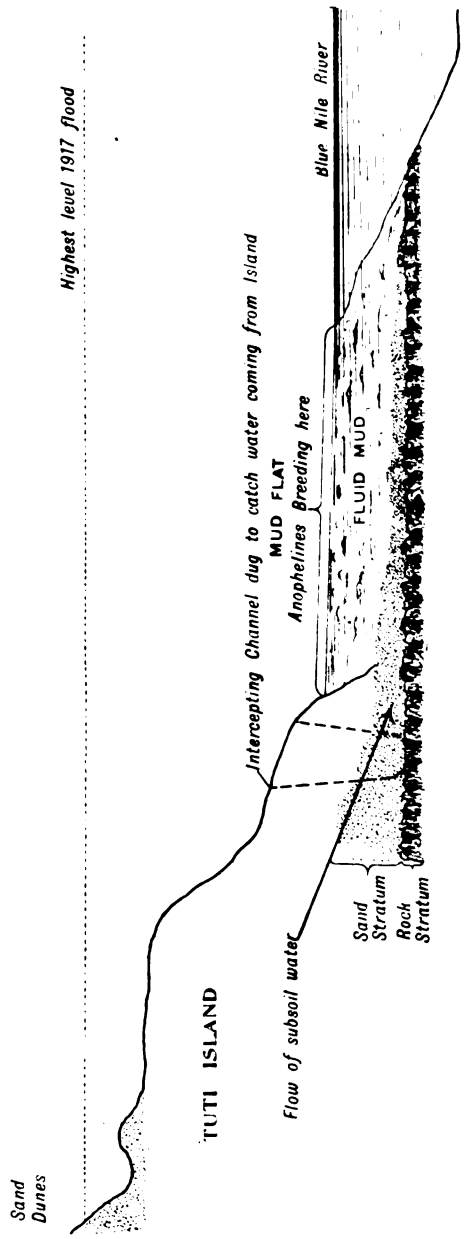

PLAN II.

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CORRESPONDENCE.

16th November, 1918.

THE REGISTRAR, COLLEGE OF PHYSICIANS,

SIR,

At a meeting of the Council of the Society of Tropical Medicine and Hygiene, held on 18th October, 1918, it was unanimously resolved, as a result of representations made by certain Fellows of the Society, to direct the attention of the Royal College of Physicians' Joint Committee, charged with the revision of the "Nomenclature of Diseases," to the following:—

1. From the point of view of tropical medicine there are a number of serious errors in the published List of Diseases.

2. There are likewise numerous important omissions, especially in the section dealing with Animal Parasites. As examples the Council may cite—

(*a*) The inclusion of two species of Simulium and *P. papatasii* (p. 234) as intermediate hosts of the malarial parasite.

(*b*) The omission of *Stegomyia pseudo-scutellaris* as a host of the filarial parasite (p. 235).

(*c*) The employment of the term "Mediterranean Fever" instead of "Undulant Fever," in view of the fact that the latter name was officially adopted by the International Congress of Medicine, held in London in 1913.

(*d*) The omission of an important intestinal parasite amongst the Flagellata, viz., *Chilomastix mesnili* (WENYON), p. 222

(*e*) The neglect to mention *Schistosoma mansoni* (SAMBON), a very important human parasite, whereas *Filaria gigas* (PROUT), which does not exist, finds a place in the list.

Many other instances have been brought to the notice of the Council. Attention may also be directed to the chapter on page 13, dealing with nomenclature. The rules there given do not correspond with those laid down in the International Rules of Zoological Nomenclature, nor with those adopted by the International Congress of Botany. Even such rules as are given are not followed in the examples cited by your Sub-Committee.

The Council of the Society of Tropical Medicine and Hygiene consider that it is regrettable that no authorities on tropical medicine were consulted on these and other important points before such a list was issued, and it trusts that when a further revision is contemplated, this oversight will be remedied.

DAVID BRUCE,

President, Society of Tropical Medicine and Hygiene.

Royal College of Physicians, London, S.W.

20th November, 1918.

DEAR SIR DAVID,

I beg to acknowledge the receipt of your letter of the 16th inst., drawing attention to errors in the revised edition of the "Nomenclature of Diseases."

I fear that nothing can be done to rectify errors in the present edition, but I will preserve your letter with the view of making alterations when that is possible.

I remain, yours faithfully,

J. A. ORMEROD,

Registrar.

Sir DAVID BRUCE, K.C.B., M.D.

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6th November, 1918.

Le President, Société de Pathologie Exotique,

DEAR SIR,

The Society of Tropical Medicine and Hygiene congratulate the Fellows of the Société de Pathologie Exotique on the happy events that have recently occurred. They rejoice that the soil of France is free from the enemy, and that the progress of freedom is now assured. They feel that in the immediate future there will be a great revival in tropical medicine, in which France and Great Britain will play the leading part, and they hope that the two Societies will always remain united by the closest bonds of fellowship, and thus cement more firmly the Entente between the two countries.

We are, yours very faithfully,

DAVID BRUCE,
President.

GEORGE C. LOW,
ARTHUR G. BAGSHAWE,
Joint Secretaries.

Institut Pasteur, rue Dutot, Paris,

Le 17 Décembre, 1918.

Le Président de la Société de pathologie exotique de Paris au Président de la Society of Tropical Medicine and Hygiene, London,

MONSIEUR LE PRÉSIDENT ET CHER CONFRÈRE,

Au nom de mes collègues de la Société de pathologie exotique, je vous remercie des cordiales félicitations que vous venez de nous adresser à l'occasion de la libération complète des régions de la France qui étaient encore occupées par l'ennemi. A mon tour, je vous adresse nos bien sincères félicitations pour la part glorieuse prise par votre Nation à la victoire qui consacre le triomphe du droit et de la liberté sur la barbarie germanique. Je souhaite que l'entente cordiale entre nos deux Nations, scellée sur les champs de bataille, s'affermisse encore dans la paix, et que pendant longtemps nos Sociétés sœurs puissent travailler, de concert et avec succès, aux progrès de la pathologie et de l'hygiène exotiques pour le plus grand bien de l'humanité.

Veuillez agréer, Monsieur le Président et cher Confrère,
l'assurance de mes sentiments tout dévoués,

A. LAVERAN,

MARCHOUX,
MESNIL,
Secretaires Generaux.

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TRANSACTIONS

OF THE

SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

FEBRUARY 21ST, 1919.

VOLUME XII. No. 4.

Proceedings of a Meeting of the Society held on Friday, February 17th, 1919, at 5.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Surgeon-General Sir DAVID BRUCE, K.C.B., F.R.S. (*President*), in the Chair.

THE MINUTE BODIES (RICKETTSIA) FOUND IN ASSOCIATION WITH TRENCH FEVER, TYPHUS FEVER AND ROCKY MOUNTAIN SPOTTED FEVER.

BY J. A. ARKWRIGHT, M.D., A. BACOT, AND F. M. DUNCAN.

From the Lister Institute.

Our work on Rickettsia has been done in connection with the War Office Committee on Trench Fever, under the chairmanship of Major-General Sir DAVID BRUCE, F.R.S., and in co-operation with Major BYAM, R.A.M.C., and the staff of the New End Hospital, Hampstead, to whom we are greatly indebted, and especially to Lieut. LLOYD, R.A.M.C., Entomologist to the Hospital.

The minute bodies to which the name "Rickettsia" was given by DA ROCHA-LIMA in 1916 are described in association with three diseases of man. They are most readily found in the invertebrate agent which transmits the disease, namely, *Pediculus corporis* or *capitis* in the cases of typhus and trench fevers, and the tick, *Dermacentor venustus*, in the case of Rocky Mountain fever. Another species is described by NÖLLER (1917) in the sheep-louse or "ked," *Melophagus ovinus*. TÖPFER (1917) claimed that Rickettsia of a distinguishable type was found in lice from cases of war nephritis, but this observation has not been confirmed.

RICKETTS (1909) was the first to describe these bodies in the blood of patients suffering from Rocky Mountain fever, and he also found them in ticks which had been fed on these patients.

RICKETTS and WILDER (1910) described very similar forms in the blood of cases of typhus and in lice taken from typhus patients. It is remarkable that they were first seen and noted in human blood films, though only small numbers have ever been found in such films, and there is so little distinctive in their appearance that they were at first not seriously recognised as competitors for the place of causal micro-organism. The attention that has been given to Rickettsia during the last three years is more dependent on its

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occurrence in large numbers in the louse or tick than on the forms seen in blood films. TÖPFER (1916) was the first to record the association of Rickettsia in lice with trench fever.

THE MORPHOLOGY AND APPEARANCE OF RICKETTSIA IN STAINED FILMS

These minute organisms resemble very small bacteria, and are usually either round, oval, diplococcal or lancet-shaped. Sometimes, when double, the two parts are separated by an unstained central segment, when they resemble a dumbbell, or they may appear like short rods with the stain concentrated at the ends, or at three or even four points in their length; one half of a diplococcal form may be much bigger than the other. They may be arranged in pairs as a short chain of three to five links, placed in a curve or L shape. The range of forms is very like that of the *B. influenzæ*. In size they vary somewhat, especially in length, but they usually appear when stained to be about 0·2 to 0·3 microns in their shorter diameter by 0.3 to 0.5 in length. They are distinctly smaller than the *B. influenzæ* or *M. meningitidis* from young cultures when these are stained side by side in the same film, and they are not so uniform in size as the latter organism.

Rickettsia is easily decolourised by alcohol in Gram's method of staining, and takes most stains feebly. It is best stained by Giemsa (one drop to the cubic centimetre) used for 16 to 20 hours.

Preliminary washing of the film with ether or fixing in ether and alcohol does not materially improve the staining by Gram's or other methods.

By Giemsa's stain the trench-fever Rickettsia is coloured, as a rule in our experience, purple or reddish purple like most bacteria. We have found it an advantage, when examining films made from the excreta of lice or from the guts of lice which contain much fresh or altered blood, to fix the film in absolute alcohol, containing 1 per cent. strong hydrochloric acid. By this means much of the débris of the red corpuscles is removed.

The forms which we have found in lice fed on human beings, or monkeys infected with typhus fever, sometimes appear slightly larger and more bacillary in shape; they also, as a rule, stain with Giemsa a redder or pinker colour than trench fever Rickettsia.

In films made from ticks infected with Rocky Mountain fever which we have been able to examine through the kindness of Dr. WOLBACH, of Harvard University, the bodies appear slightly larger, and more lancet-like or bacillary in shape.

We have found that the best routine method of examining lice which have been fed on trench fever patients with a view to determining whether they are infected with Rickettsia is to make a film of the excreta of the lice. This appears to have been very little done by other observers, who have worked almost entirely by cutting sections of lice or making films of their intestinal contents We have not so readily found Rickettsia in the excreta of lice which have fed on typhus patients.

Examining the excreta enables observations to be made on a box of lice for a number of consecutive days without destroying the lice. The lice with which we have worked have been kept in boxes with a gauze cover, through which they feed and through which the excreta can be daily shaken out.

In films shewing Rickettsia, whether of excreta or single lice, the micro-organisms are often seen in enormous numbers, and may be in masses which appear to consist of nothing else. Often they are seen embedded in flakes of semitranslucent material, which is, perhaps, mucus or part of a disintegrated cell. Under these latter conditions the Rickettsia stains well, and shews a very definite outline lying in the feebly stained matrix. In sections of lice the same appearance is seen in parts of the gut where the contents are reduced in bulk, and the blood débris has almost disappeared.

The most distinctive characters of Rickettsia in lice and their excreta are the size, number and staining properties, besides the shape and arrangement. The last two characters appear to us to be insufficient alone for the identification of these bodies when they are few in number and scattered singly in a film. Granules of similar size and shape are often to be seen in films of the excreta of normal lice or of healthy blood which has been for some time in the incubator; they can usually be recognised by their shadowy appearance, feeble staining and indefinite margins. It is not intended to convey the impression that the Rickettsia is always easy to recognise in films of louse excreta or gut contents. Sometimes a stained precipitate or deposit of stain may cause much uncertainty.

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However, in a very large proportion of infected louse excreta, or in infected lice, the numbers and typical appearance leave no room for doubt. We have always recorded the result as negative in doubtful cases.

REGULARITY OF THE ASSOCIATION OF RICKETTSIA IN LICE WITH TRENCH FEVER IN MAN.

Our work has been chiefly done with trench fever, but we have made a few observations on typhus, which will be mentioned later.

We have been much struck with the great regularity with which Rickettsia appears in the excreta of lice when these are examined daily after feeding on a trench fever patient. The first appearance is, as a rule, eight to ten days after the first infecting feed. The occurrence is extraordinarily regular if the lice are fed twice daily on the same infected man, beginning on the first or second day of the disease. A like result usually follows feeding on an infected man for one day only, and subsequently on a healthy man. Lice do not, however, necessarily become infected with Rickettsia every such day on which they feed, and not all of those fed two or three times shew Rickettsia.

These results are well shown by reference to an experiment carried out on two volunteers—Purton, who was experimentally infected with trench fever, and a healthy man, Elliot. Purton was inoculated with the excreta of infected lice.

This experiment was carried out with great care and no little trouble at the New End Hospital, Hampstead, by Lieut. LLOYD, to whom we are very much indebted, and the lice and excreta were examined at the Lister Institute by the present writers.

A fresh box of about fifty healthy lice was fed on Purton every day, and subsequently fed daily on Elliott. All the lice used were from the same healthy stock, and each box contained lice of similar age. This course of feeding was begun on the day on which Purton was inoculated and was continued for a fortnight, *i.e.*, through six days of incubation before Purton became feverish until the eighth day of Purton's disease. It was preceded by feeding fifty lice in another box on Purton for the five consecutive days immediately before the inoculation, as a control to shew that he was not in a condition to infect lice with Rickettsia before the inoculation. In addition, two boxes were fed on Purton on the first day of his illness, and twice daily on him subsequently. The lice were contained in gauze-covered boxes, and during feeding were bandaged on to the forearm for thirty minutes.

All the lice were kept in an incubator at 30° C. between their feeds

In this experiment twenty boxes of lice were used and examined daily for three weeks.

TABLE I.

Date.	Fed on	No. of Box.	Duration of feeding.	Appearance of R. in excreta.	Duration of R. in excreta.	Proportion of infected lice.
Control						
22 x. to 20 xi.	E	260	33 days	0	0	nil
21 to 25 x.	P	261	4 ,,	0	0	nil
26 x.	P inoculated	262	1 ,,	0	0	nil
27 x.	P	263	1 ,,	0	0	nil
28 x.	P	264	1 ,,	0	0	nil
29 x.	P	265	1 ,,	0	0	nil
30 x.	P	266	1 ,,	0	0	nil
31 x.	P	267	1 ,,	0	0	nil
1 xi.	P	268	1 ,,	twelfth day	2 days	0 in 6 = 0 %
2 xi.	Onset of fever					
2, 3 xi.	P	269	2 ,,	0	0	nil
2 xi.	P	270	1 ,,	0	0	nil
3 xi.	P	271	1 ,,	ninth day	6 days	1 in 37 = 2·7 %
4 xi.	P	272	1 ,,	0	0	nil
5 xi.	P	273	1 ,,	eighth day	14 days	6 in 27 = 27 %
6 xi.	P	274	1 ,,	ninth day	12 ,,	3 in 24 = 12·5 %
7 xi.	P	275	1 ,,	eighth day	20 ,,	12 in 41 = 28·6 %
8 xi.	P	276	1 ,,	seventh day	15 ,,	1 in 44 = 2·3 %
9 xi.	P	277	1 ,,	eleventh day	15 ,,	9 in 28 = 32 %
2 to 18 xi.	P	258	16 ,,	ninth day	more than 8	——
2 to 26 xi.	P	280	23 ,,	ninth day	,, ,, 11	13 in 16 = 81 %

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Table I. and Chart I. shew the results obtained by examining films made from the excreta in the different boxes daily.

It is seen that the excreta of the lice fed during the four days before inoculation and from the day of inoculation up to the fifth day of incubation never shewed Rickettsia. On the other hand, seven of the ten boxes of lice fed on Purton on one day only each from the sixth day of incubation to the eighth day from the febrile onset did shew Rickettsia after the lapse of seven to twelve days from their feed on Purton. Also the excreta from both boxes, Nos. 258 and 280, fed twice daily on the infected man shewed Rickettsia on the ninth day from the first feed on him.

To sum up, most of the boxes of lice fed on this trench fever patient early in the disease became infected with Rickettsia after an interval which may be called the incubation period in the louse, but the lice in similar boxes fed on the same man before his illness or on a healthy man did not excrete Rickettsia. When the results of feeding lice on different days are examined in detail it is seen that those feeding on some days during the illness became infected, but those feeding on other days did not. Moreover it was not necessary that the patient should be febrile in order that the lice should become infected; indeed in this series and in some other experiments the lice appear to have been more readily infected after than during the first days of fever. It is interesting also that the box of lice fed on the day before the rise of temperature became infected.

Another interesting point comes out on examining this series, namely, that the lice in some of the boxes were more heavily infected than in others. This was shewn by differences in the proportion of lice found infected when films were made from single lice, and also by the fact that the excreta from two of the boxes shewed Rickettsia for only a few days, whereas most of the boxes which yielded infected excreta continued to do so as long as they were examined.

Thus, in box 268, fed on the day before the onset of fever, Rickettsia were found for two days only, and in another box, 271, only for six days. This latter box was fed on the second day of fever, and thus between two boxes, 270 and 272, neither of which ever yielded any Rickettsia. There is also a difference in the incubation in the louse before the Rickettsia appears; those boxes which are most infected appear to shew a shorter incubation as a rule.

A short period during which a box is infected is held to shew that only a small number of lice in that box were infected, and that these few died out early, leaving no further source for Rickettsia. The differences in the results of feeding on different days of illness are interpreted as indicating variation in the number of the Rickettsia in the peripheral circulation. Sufficient experiments have not been made to enable us to state definitely that the peripheral blood is always less infective for lice during the first two or three days of the disease, but such evidence as we have is in favour of this view. The two boxes fed daily became infected before the others, *i.e.*, both on the ninth day of the illness, and became the most heavily infected as shewn by the very large number of Rickettsia in the excreta and the high percentage (81 per cent.) of infected lice found in box 280. It is not necessary for our present purpose to recount further experiments in detail. It is enough to state that when lice are fed twice daily on a trench fever patient during the first week of his illness (and often much later), Rickettsia invariably appears in a proportion of the lice and their excreta after an interval of five to fourteen days, or rarely somewhat longer from the first infecting feed, if the lice are kept at a temperature of 27° to 32° C. between the feeds.

It has also been shewn that the excreta of lice fed in such a manner become infective for man after an interval of some days (about a week) from the first infecting feed.

For further details we must refer to the paper by Major BYAM, R.A.M.C., and the Staff of the New End Hospital, Hampstead, read before this Society in May, 1918, and to a note by us in the *British Medical Journal*, of 21st September, 1918, and a more detailed account of our work in the *Journal of Hygiene* of April, 1919 (ARKWRIGHT, BACOT and DUNCAN, 1919).

ASSOCIATION OF RICKETTSIA WITH TYPHUS.

With regard to Rickettsia and typhus fever we have done comparatively little work, and for the evidence that this organism is constantly associated with the disease we are

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mainly indebted to DA ROCHA-LIMA (1916), TÖPFER and others. We have, however, made a few observations, which are summarised below:—

1. Amongst a number of lice, *Pediculus corporis*, which had been fed on typhus fever patients, we found a louse containing large numbers of bodies closely resembling the Rickettsia bodies of trench fever.

2. Twenty lice from the same source were inoculated subcutaneously and intramuscularly into a *Macacus rhesus*, No. 14, which developed an illness with fever after twelve days' incubation, and died on the twenty-second day from the date of the infection; no cause of death was found macroscopically post-mortem.

3. Another *M. rhesus*, No. 16, was inoculated subcutaneously and intramuscularly with 0·25 c.c. on 9th January, and with 3·5 c.c. on 10th January, of the blood of No. 14, taken on the seventh and eighth days of disease respectively.

No. 16 developed a well-marked febrile illness after five days' incubation.

4. A large number—about 150—monkey lice, belonging to the genus Pedicinus (probably *P. longiceps*, PIAGET) were taken from No. 14 after death, and were then emulsified in physiological saline solution and inoculated, on 13th January, subcutaneously and intramuscularly into No. 17. Also 20 to 30 pediculi fed on No. 14 from 9th to 11th January were fed on No. 17 from 13th to 21st January. This latter monkey, *M. rhesus*, had fever, beginning on 21st January, after seven days' incubation. It became very ill, and was killed on the tenth day of illness.

5. Two guinea-pigs inoculated with emulsions of spleen and supra-renal of No. 14 respectively shewed well-marked febrile reactions after nine and ten days.

6. Pediculi were fed on monkey No. 14 on the last (seventh, eighth, ninth) days of illness when the temperature was falling. The excreta from the box of lice examined on the sixth day from the first possibly infecting feed shewed Rickettsia bodies. The lice had been fed after the death of No. 14 on a fresh monkey, No. 17. Considerable difficulty was found in keeping pediculi alive which were fed on these monkeys

7. Pediculi were also fed on monkey No. 17 during the febrile period. Of four of these lice examined on the sixth to the ninth days from the first infecting feed, three were found to contain Rickettsia, or bodies which we take to be the Rickettsia, of typhus fever.

8. Lice (Pedicinus) native to the monkey, were taken from Nos. 14, 16, 17, and also from three normal control monkeys, and films were made from the alimentary canals of single lice and examined for Rickettsia. The results are shewn in Table II.:—

TABLE II.

INFECTION OF MONKEY LICE (PEDICINUS SP.) WITH THE RICKETTSIA ASSOCIATED WITH TYPHUS.

Monkey.	Day of Disease.	No. of Lice examined.	No. shewing Rickettsia.	Percentage.
No. 14	7th to 10th	35	6	16 %
No. 16	9th to 17th	26	1	4 %
No. 17	8th to 9th	32	5	16 %

Control—Lice taken from Normal Monkeys..

Monkey.	No. of Lice examined.	No. shewing Rickettsia.
No. 10	38	0
No. 29	20	0
No. 31	17	0
	75	0

The Rickettsia which we have seen in lice, which have fed on typhus patients and monkeys, have closely resembled those seen in connection with trench fever, but so far as our observations go the following differences have been noticed:—

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1. We have only once found the Rickettsia of typhus in the excreta of lice, though we have often looked for them.

2. The bodies found in connection with typhus stain a redder colour with Giemsa than those of trench fever, and sometimes appear slightly larger, and are more often lancet-shaped or like segmented bacilli in form.

Sections of Lice.

Da Rocha-Lima (1916, 1917) and others have described Rickettsia bodies in very large numbers in the alimentary canal of lice fed on typhus and trench fever patients. It is stated that in microscopic sections these bodies can be seen within the epithelial cells of the gut in typhus but not, according to da Rocha-Lima, in trench fever.

In some of our sections of Pediculi from trench fever patients, and of Pediculi and Pedicini taken from typhus patients and monkeys, Rickettsia bodies are to be seen in enormous numbers lying close to the epithelium in the lumen of the midgut, and also in groups scattered through the altered blood which constitutes the gut contents.

In the material so far examined we have not seen definite invasion of the epithelial cells.

Rickettsia in Rocky Mountain Spotted Fever.

Ricketts (1909) and more recently Wolbach (1916 and 1918) have described minute organisms in very large numbers in the alimentary canal and tissues of the tick. Wolbach also describes their presence in sections of the skin of human patients and in the skin and testis of infected guinea-pigs.

Through Dr. Wolbach's kindness we have been able to see films from the tick and sections of infected mammalian tissue. The organisms in the tick appear slightly larger than the Rickettsia of trench fever and typhus, and are usually longer and more lancet-shaped.

Discussion.

The criticisms of da Rocha-Lima's work and of his interpretation of his observations have been brought along two main lines.

1. It has been maintained by some observers that these Rickettsia bodies are not micro-organisms, but granules of precipitated or coagulated protein, such as are found in old blood cultures.

2. It has been emphasised that these bodies were found by him and others in lice taken from persons who were free from any known contact with typhus.

3. A further criticism has been based on the earlier experiments of Nicolle, Conor and Conseil (1910), which seemed to shew that the virus of typhus would pass through a bacterial filter. The same argument against Rickettsia bodies being the virus of trench fever follows from the filtration work of the American Red Cross Committee on Trench Fever (1918).

The first criticism that the forms seen are not micro-organisms is upheld or discounted largely according to the personal opinion of the observer based on the microscopic appearance and the technique used in preparing the specimens. This criticism is most effective when the observer has himself made and stained his own films. However, if the view that the bodies are not a living virus is maintained it is necessary to account for their presence in infected lice and their excreta and not in normal lice nor in their excreta.

The second criticism that these bodies are found apart from typhus was to a great extent removed by the observation that similar forms occurred in lice which had fed on trench fever patients. Trench fever has been so frequent and widespread during the past few years in the war areas that it is not surprising that Rickettsia has been found in connection with persons other than typhus patients.

There still remain a few instances of lice from apparently healthy men in whom large numbers of Rickettsia bodies have been found by continental observers. Da Rocha-Lima (Munk and da Rocha-Lima, 1917), called the bodies in trench fever lice *R. quintana* and those in normal lice *R. pediculi*, to distinguish them from the bodies associated with

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typhus, for which he retained the name *R. Prowazeki*. NICOLLE, BLANC and CONSEIL (1914) recorded finding the bodies described by SERGENT in 5 per cent. of the lice taken from persons in a district quite apart from any epidemic focus.

BRUMPT (1918) found Rickettsia in a high percentage of lice taken from seven prisoners of war at Rennes, who were apparently in good health, but he apparently only attempted to exclude contact with cases of typhus fever as he does not mention trench fever in his paper.

In London we have not so far found Rickettsia in lice from healthy civilians, and we attribute this to the practical absence of trench fever and typhus from the civilian population.

The claim that typhus fever virus is filterable was not confirmed by GOLDBERGER and ANDERSON (1912) and other workers. With regard to trench fever, four experiments on volunteers have been made by the War Office Committee with filtered emulsions of the infective excreta of trench fever lice. After filtering through a Chamberland F the filtrate failed to reproduce the disease in man.

It seems probable that the varying results under different circumstances are due to the size of the particles composing the virus. The Rickettsia bodies appear to be about 0·2 to 0·3 microns in diameter, *i.e.*, of such a size that during three hours' filtering some would occasionally pass through a filter which would keep back the *B. typhosus*, which was the test bacillus used to control the permeability of the filter by the American Committee.

Although this paper was written before its appearance, it is impossible to pass by without mention the paper recently published by ROSE BRADFORD, BASHFORD and WILSON (1919) in which it is claimed that the virus of trench and typhus fevers, among other diseases, has been cultivated in artificial media after passage through a Berkefeldt, and that these diseases have been reproduced by inoculation of the cultures. The work of these writers has not yet been published in sufficient detail to judge of its bearing on the rôle of Rickettsia in the diseases with which we are now concerned.

It seems that since the bodies which they describe are approximately of the same size as Rickettsia, the two forms may possibly be identical and if both are truly micro-organisms that their identity is probable

A difference in the two descriptions so far given is that while Rickettsia is by all observers stated to be readily decolourised by Gram's stain, the bodies of ROSE BRADFORD, BASHFORD and WILSON are Gram-positive. We have never seen Rickettsia retain the stain by Gram's method, although washing the film with ether has been tried as recommended by the last named workers.

It is perhaps unnecessary and premature at the present time to discuss the position of Rickettsia in a systematic classification. PROWAZEK and DA ROCHA-LIMA tend to the view that they are protozoa (Strongyloplasmata or Chlamydozoa). RICKETTS, WILDER, SERGENT, FOLEY and VIALATTE (1914) and TÖPFER appear to regard them simply as bacteria. WOLBACH apparently thinks that the micro-organism of Rocky Mountain fever which is found in the tick passes through slightly different forms in different phases of development. In any case it seems useful to retain the name Rickettsia for the present to denote the group of very small organisms including those of typhus and trench fevers, which, though to be seen in very small numbers in the blood of the infected mammalian host, are much more readily found in the invertebrate host which transmits the disease, and then are massed in enormous numbers in the alimentary canal and sometimes elsewhere.

CONCLUSIONS.

Trench Fever.—The evidence for the association and causal connection of Rickettsia with trench fever may be summarised as follows:—

1. The organisms are constantly present in lice and the excreta of lice which have fed on trench fever patients, and are infective for man.

2. Their presence in these infective materials, usually in enormous numbers, corresponds to the minute dose, *e.g.*, the contents of the gut of one louse about 1 milligramme in weight, or a fraction of a milligramme of excreta required to infect a man.

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3. Their appearance in the louse after an incubation period, usually of eight to ten days, which agrees roughly with the incubation period of the virus in the louse (five to twelve days). The virus was shewn to be present in these cases by human experiment.

4. The size of the Rickettsia is such that it would usually be held back by a bacterial filter, but may occasionally pass a filter which retains such bacteria as *B. typhosus*, or *B. prodigiosus*, and the virus also has been found to sometimes pass through a porcelain filter and sometimes not.

Typhus Fever.— The evidence of the connection of typhus fever with Rickettsia is of the same kind as in the case of trench fever, but the presence of the infecting virus can be shewn by the inoculation of monkeys and guinea-pigs. Our own observations lead us to the following conclusions:—

1. The Rickettsia of typhus does not appear in the excreta of lice with the same regularity as Rickettsia of trench fever.

2. Monkey-lice, *Pedicinus longiceps* (?) taken from a monkey infected with typhus on the seventh to the eighth day of the disease have been shewn to contain Rickettsia and probably typhus virus. Pedicini from monkeys which have not been inoculated have never, so far, been found to contain Rickettsia (75 lice examined).

3. Our comparatively few observations confirm the work of previous observers in the following particulars:—

(*a*) Pediculi fed on typhus patients may contain both the virus of typhus fever and Rickettsia.

(*b*) Both guinea-pigs and monkeys react with fever to the inoculation of typhus virus, whether in blood or lice or emulsions of organs taken post-mortem from animals.

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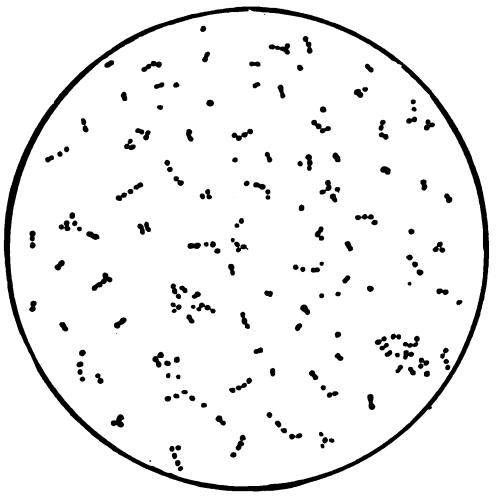

From excreta of lice (*Pediculus humanus*) fed on Trench Fever case.

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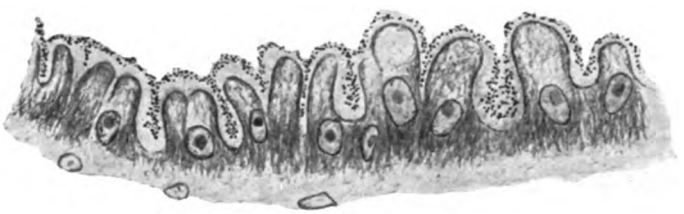

From section through gut of louse (*P. humanus*) fed on Trench Fever case.

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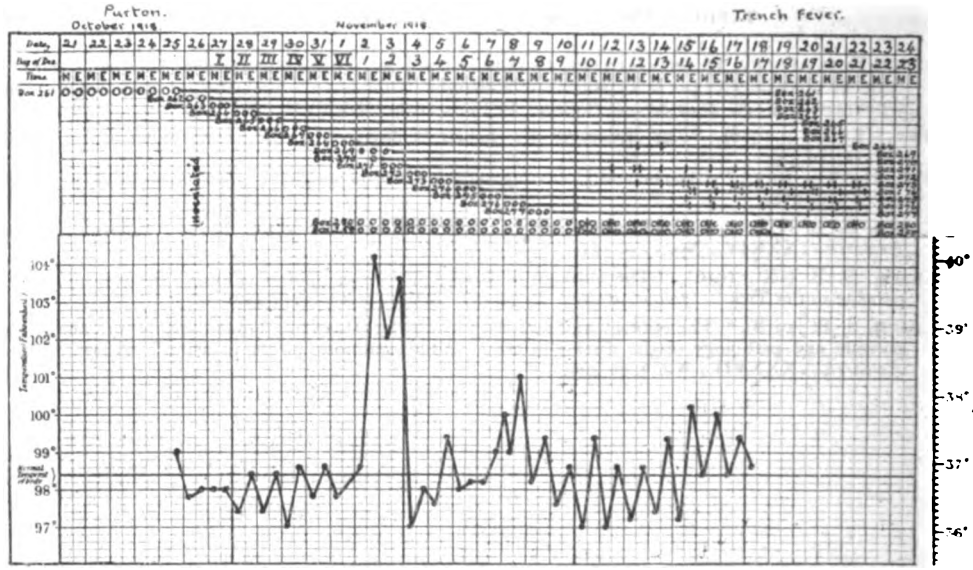

CHART I.

The lower part of the Chart shews the temperature of the volunteer Purton, who was inoculated with trench fever. Each of the horizontal lines (261 to 277, 280 and 258) in the upper part of the Chart represents a box of lice, and each sign O indicates that the lice in that box were fed on Purton; the cross-marks, +, + +, on these lines shew the days on which the Rickettsia was found in the louse-excreta. The lines with no crosses represent boxes from which Rickettsia was never obtained. Boxes 280 and 258 were fed on Purton twice daily; the boxes 262 to 277 were only fed on Purton on one day each; box 261 was fed on Purton on the four days immediately before he was inoculated.

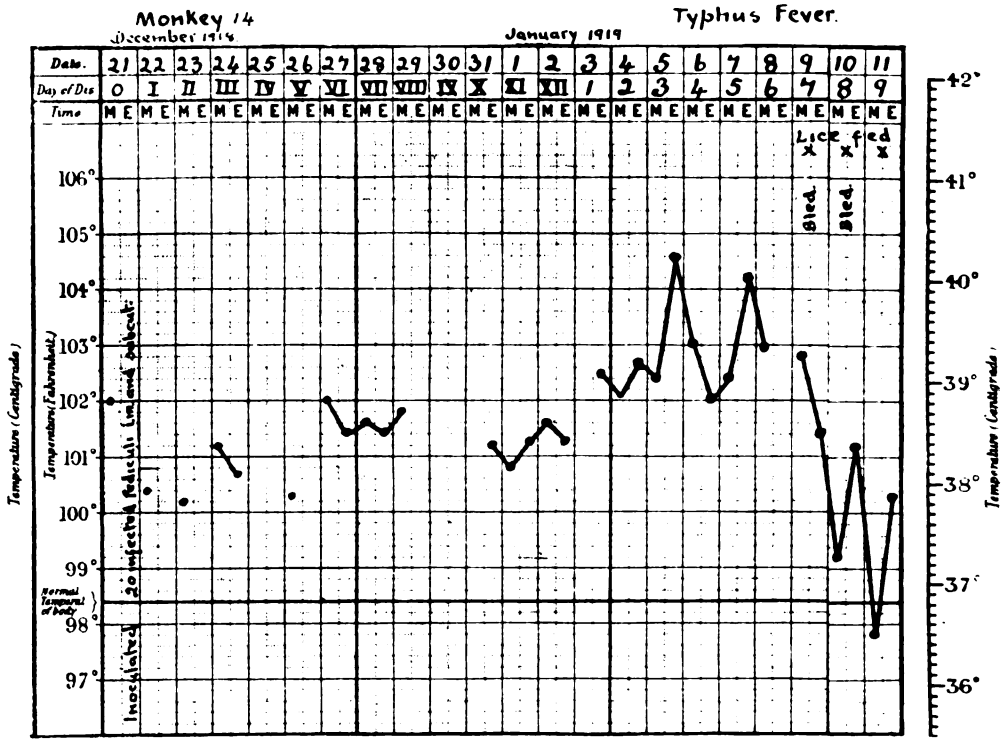

CHART II.

Monkey No. 14, *Macacus rhesus*, inoculated subcutaneously on 21st December, 1918, and intramuscularly with an emulsion of 20 *Pediculus corporis* from boxes which had fed on five human cases of typhus fever between the 5th and 15th Dec., 1918, on the 6th–11th and 4th–11th, 9th–15th and 11th–16th days of the fever respectively.

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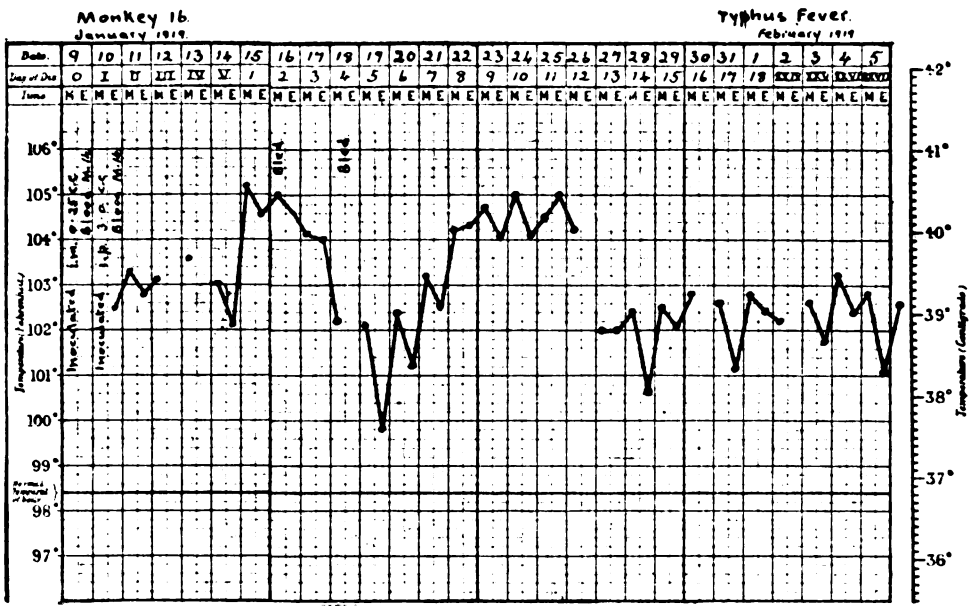

CHART III.

Monkey No. 16, *Macacus rhesus*, inoculated on the 9th January with 0·25 c c. of blood, and on 10th January with 3·5 to 4 c.c. of blood from Monkey No. 14, taken on the 7th and 8th days from the onset of the disease in Monkey No. 14.

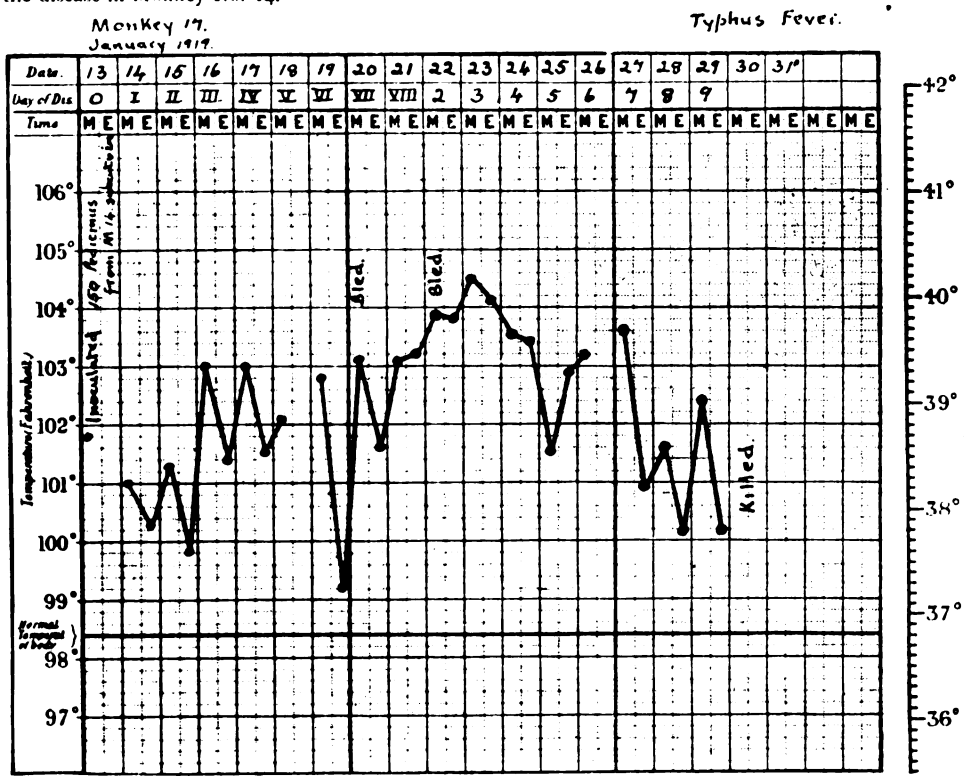

CHART IV.

Monkey No. 17, *Macacus rhesus*, inoculated subcutaneously and intramuscularly with an emulsion of about 150 lice (*Pediculus*) taken from Monkey No. 14 on the 12th Jan. after death. Pediculi which had been fed on Monkey No. 14 on the 9th and 10th Jan. were also fed on Monkey No. 17 daily from the 13th to the 29th Jan.

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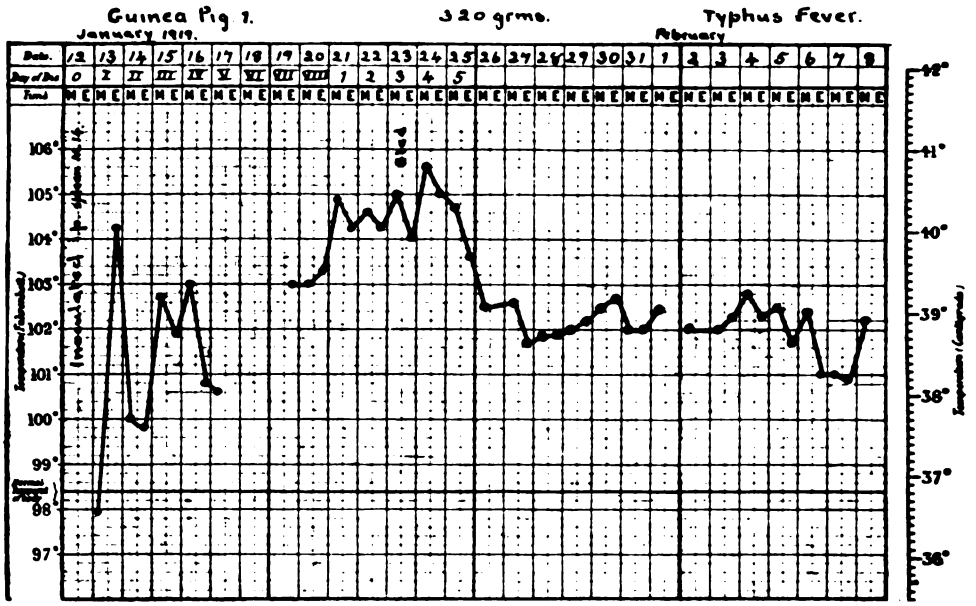

CHART V.

Guinea-pig No. 1, inoculated intraperitoneally with an emulsion of the spleen of Monkey No. 14.

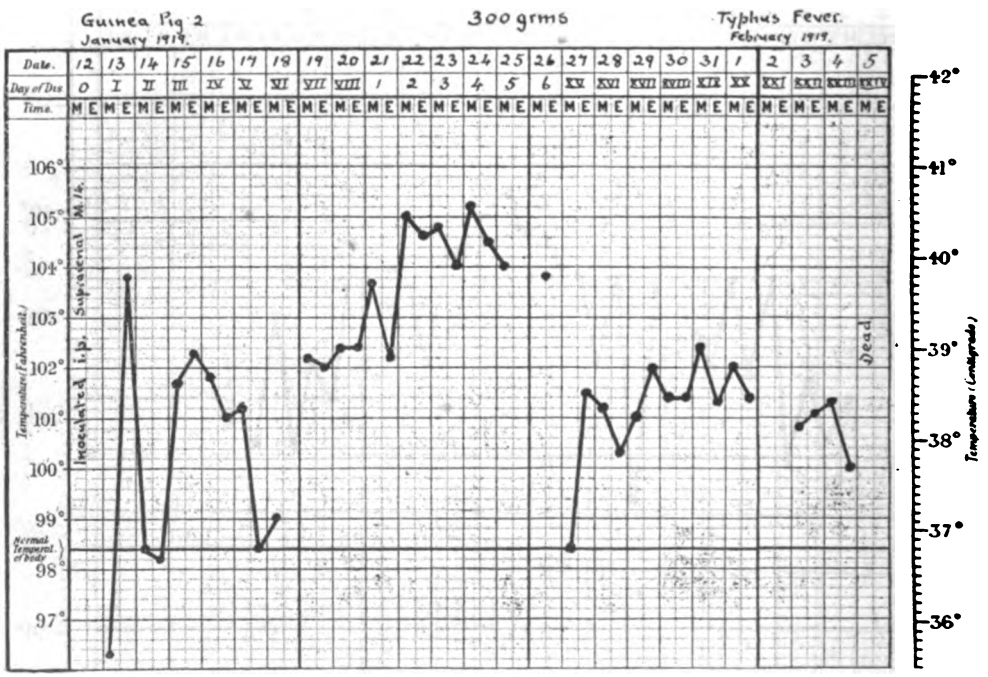

CHART VI.

Guinea-pig No. 2, inoculated intraperitoneally with an emulsion of the suprarenal of Monkey No. 14.

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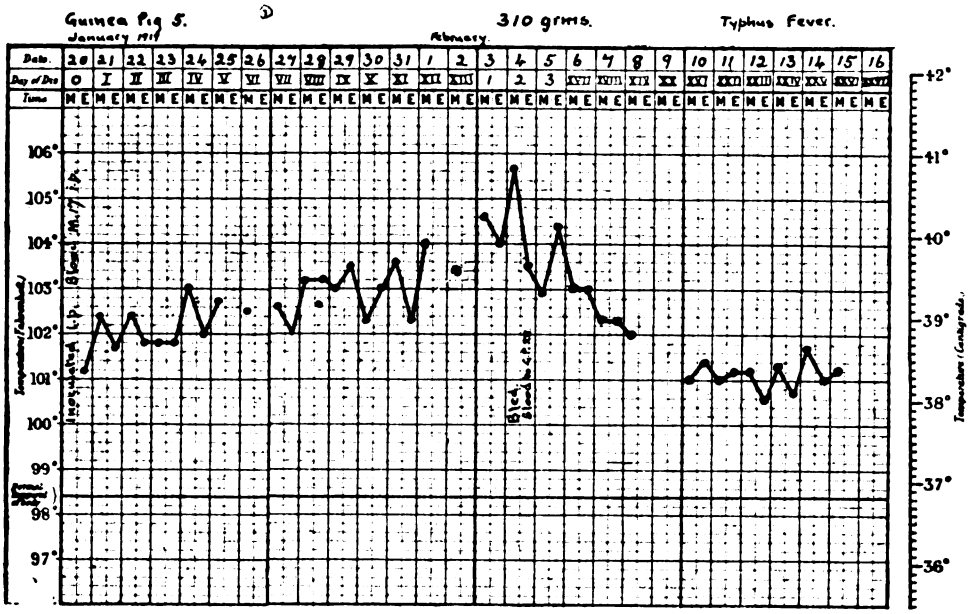

CHART VII.

Guinea-pig No. 5, inoculated intraperitoneally with 0·5 c.c. of citrated blood from Monkey No. 17.

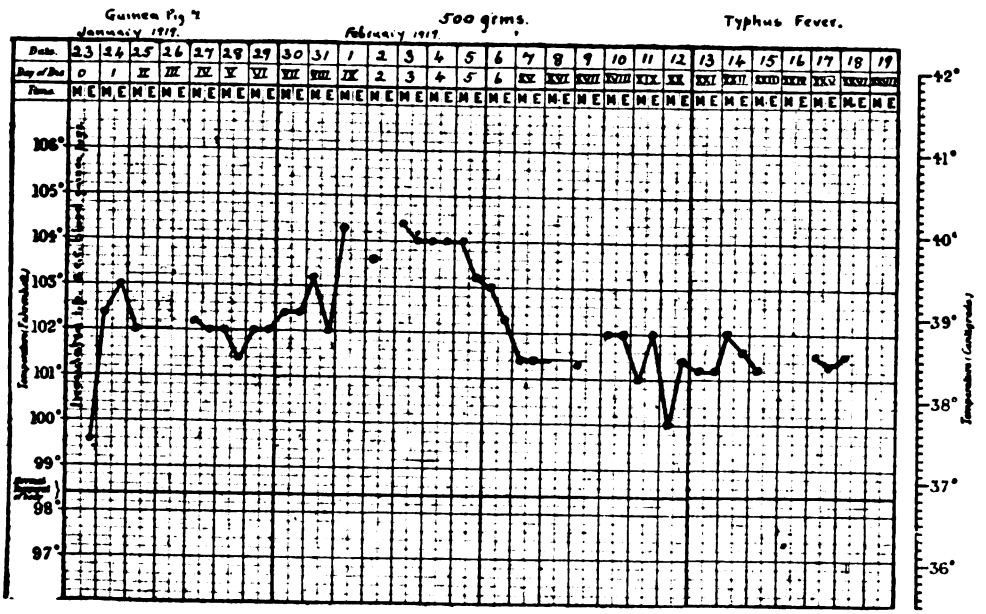

CHART VIII.

Guinea-pig No. 7, inoculated intraperitoneally with about 2 c.c. of citrated blood from Guinea-pig No. 1, taken on the third day of the fever.

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THE PATHOLOGY AND EPIZOOTIOLOGY OF LOUPING-ILL (DISEASE OF SHEEP). WITH SPECIAL REFERENCE TO CHROMATIN BODIES IN THE WHITE CORPUSCLES

By Sir STEWART STOCKMAN, *Chief Veterinary Officer Board of Agriculture and Fisheries.*

I have been engaged in the investigation of louping-ill for several years past. It is a seasonal disease and, therefore, one with regard to which investigation can only be pressed during a limited period of each year when it is in evidence. I will not attempt in a short space of time to go into every point which has been the subject of investigation, and I propose to limit myself to the results regarding what might be referred to as the fundamental points, making special reference to certain bodies, which I have provisionally called "chromatin bodies," present in the cells of lymphatic glands and of the blood of animals affected with the disease, as these may possibly occupy the important position of being the causal agent (full papers published in *Journal of Comp. Pathol. and Therap.* 1917 and 1918).

Some years ago I was able to shew by experimentation that British ticks could be infected by the piroplasm of the redwater of this country, and that this invertebrate host of the causal parasite could in a subsequent stage of its life cycle transmit the disease to other cattle, and further, that British ticks, given the opportunity, could become infected with, and be transmitters of, the tropical form of redwater, although the latter is caused by a different species of piroplasm. In pursuing these investigations, which involved innumerable examinations of blood from different animals, I also became aware that there exists an inoculable trypanosomiasis of British cattle, but the questions of whether it is insect-borne, and, if so, what insect is responsible, have not yet been solved. The point of these remarks in bringing to your notice another tick-borne disease of this country—a disease of sheep known popularly as "louping-ill" and "trembling" is to call attention to the following:—

As the investigation of diseases of animals proceeds it becomes more and more clear that diseases which are carried by invertebrate hosts, in which it is essential that the causal agents undergo part of their life cycle, are not by any means exclusively tropical or subtropical. It also appears probable that certain diseases classed as tropical could be established in this country.

Louping-ill causes very considerable losses on certain sheep farms, principally in the north of England and the south of Scotland, but it is by no means confined to these areas It has been the subject of investigation by several pathologists working singly, and by a number of committees appointed by Government departments and agricultural societies.

Various claims have been made to the discovery of the cause of louping-ill, all of which have rested upon isolating a microbe from some part of the body of a sheep dead of the disease, and causing illness or even death with cultures of these microbes by inoculating them to other sheep. Such a method of investigation, particularly when applied to diseases of sheep, invites error, because in that species of animal cadaver bacteria invade the tissues

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with such extraordinary rapidity that they may be found in parts distant from the intestines in a few hours; indeed, transudates in the peritoneal cavity may even be invaded before death. None of the claimants to the discovery of these alleged causal microbes seem to have followed a methodical and obviously correct system of examining and inoculating the tissues or fluids of affected sheep obtained while alive, or from those which were slaughtered instead of being allowed to die, although recognising the nervous characteristics of the disease, portions of nerve tissues were frequently employed with negative results. Further, although the question of ticks being a factor in the causation of louping-ill has been discussed from time immemorial, both by farmers and by scientists, it seems always to have been dealt with in a somewhat partisan spirit by both, and was never submitted to investigation in the scientific sense, most preferring to accept it doubtfully, or reject it, entirely on the circumstantial evidence as it appealed to them individually. It is perhaps not surprising that the circumstantial evidence often gave rise to contradictory views, as the disease was originally identified by, and received its names from, farmers, the almost inevitable result being that different disease conditions were often grouped under the same name, and the epizootiological data were divergent.

Among the preliminaries which had to be undertaken in this investigation were:—

1. To select for the initial work only those cases in the field which might be looked upon as having a clinical or post-mortem entity; these were all collected from notoriously infected farms.

As regards macroscopic lesions the matter turned out to be simple in one sense, as it eventually became evident that the only really constant lesion was swelling and œdema of some of the lymphatic glands with sometimes congestion; this, however, in no way explained the symptoms. The possible bacteriological aspect of the disease was settled as regards microscopically visible microbes at a comparatively early stage of the investigation by finding that the fluids, tissues, and juices of animals slaughtered in various stages of the disease instead of allowing them to die were invariably sterile.

As regards clinical symptoms, it was found after careful sifting that cases could be grouped under three heads:—

(*a*) The animal may be found down in a senseless and completely comatosed condition with the head drawn round towards the shoulder, having lost all power to maintain itself on its legs, although the legs may be capable of performing paddling movements when the patient is laid on its side.

(*b*) The patient may be found down but quite sensible, the anterior, or posterior, or both extremities, being paralysed.

Along with these basic nervous symptoms others, such as general trembling, twitching of the muscles of the jaw, and frothing at the mouth, may be seen.

(*c*) It was said by many shepherds and flock-owners, and it agreed with my own observations, that every sheep reared on, or brought on to, louping-ill pastures passes through an attack of the disease in some form, and, if it survives, it becomes highly resistant to further attacks, at least in a dangerous form. Many sheep, however, never show objective symptoms visible to the ordinary observer, but shepherds and flock-masters of experience can pick them out as affected. This means that louping-ill may assume the form of a more or less slight indisposition.

(*d*) The indisposition may be somewhat greater than in (*c*) and the animal may lie down continuously apart from its companions, only rising at intervals to move a short distance, and then lie down again, or it may only move when chased.

For purposes of investigation, only cases shewing distinctive paralytic symptoms were chosen in the first instance, the object being to avoid as far as possible the inclusion of other diseases. When a lesion, such as an abscess, was found in a position in which it might possibly have caused paralytic symptoms, the case was also rejected. Following this method of eliminating more or less doubtful material it became apparent that a defined diseased condition existed, having for its distinctive symptoms those described under (*a*) and (*b*). On histological examination of the brains and spinal cords of such cases they shewed more or less definite lesions, which I need only refer to generally as those of

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myelo-encephalitis. I may here add that the mild cases slaughtered for examination a a later stage of the inquiry shewed similar lesions in a lesser degree, and that the experimental cases to be referred to shewed the same lesions.

2. It seemed important to sift, analyse, and make further observations in connection with the various statements regarding the circumstantial evidence for and against the tick as a factor. This resulted in the adoption of the following definite conclusions :—

(*a*) Ticks are always present on the pastures where true louping-ill is present, although it has been asserted that this is not so. It is true, however, that the disease may be unknown on land infested by ticks, but this may only mean that the ticks on such pastures have never had the chance of becoming infected.

(*b*) That louping-ill is a seasonal disease occurring chiefly in April, May and early June and to a less extent in autumn, and that these times are times when ticks in various stages, but ready to feed, are most plentiful.

Let us return for a moment now to the most constant macroscopic lesion, namely, the œdematous lymph glands. On cutting into these a very thick creamy juice was found in the medullary portion. Smears made from this creamy material when stained by Giemsa stain shewed the chromatin bodies to which I will later refer more particularly. The condition of the glands and the presence of these bodies suggested that inoculation with this material might be followed by interesting results. It may be said here, however, that material from the œdematous glands, the blood, and other tissue juices was all examined carefully by dark-ground illumination for motile bodies with negative results; the only motile bodies found being gyrating particles indistinguishable from those seen in normal blood and tissue juices.

An affected lamb (21) was sent up alive to the laboratory from an infected farm which was being used as a field observation station, On arrival it shewed the disease in one of its distinctive forms, its hind legs being completely paralysed. It was killed by bleeding. All the organs and tissue juices proved sterile on media. Microscopical lesions of the cord (examined at a later date) were evident; the cells of the œdematous glands and the blood shewed chromatin bodies. (The lesions of the cord were illustrated on screen.)

Experiments 52-55—Two ewes and two lambs were inoculated subcutaneously with 5 per cent. of an emulsion from the œdematous glands of the above lamb. One ewe (12a) became distinctly ill on the eleventh day and died paralysed on the thirteenth. The other ewe (14) on the twelfth day had become dull, listless and disinclined to move, but it had apparently recovered on the sixteenth day. One lamb (11a) became ill on the ninth day and died paralysed on the twelfth. The other lamb (13) became ill on the ninth day and died paralysed on the twelfth. The three animals which died shewed typical lesions in the cord and chromatin bodies were present in the swollen glands. The bodies were present in blood smears from all four inoculated animals, one of which did not die.

(These are only selected experiments for illustration, others were shewn on the screen.)

These results were not accepted as conclusive at this stage because of certain apparent failures at the time to carry on the disease by inoculation in series.

Tick Experiments.

The sheep tick which prevails exclusively on infected farms is the *Ixodes ricinus*. I have made many observations on the habits of this tick as seen on louping-ill pastures, but with only a few of these need I take up your attention. The *I. ricinus* is known as a thrice sucking tick, which leaves its host and moults on the ground each time after engorgement, that is to say, it requires three vertebrate hosts for its full life-cycle. The prevalence of this or that stage on the pastures, then, at a particular time of the year depends to some extent on whether its hosts are available to enable it to pass from one stage to another. On these sheep pastures larvæ, nymphæ and adults are exceedingly plentiful in April and May. All forms are again found in August, September and October, but not so plentifully. These, as already mentioned, are the louping-ill seasons, but the main damage is done in the spring, because of the advent of new lambs which provide fresh and suscep-

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tible fuel for the disease, most of the old stock having become highly resistant by the autumn. If sheep of any age from clean farms be introduced at these seasons, however, the losses may be enormous. Experiments were carried out with ticks which in some previous stage had fed upon an infected sheep in the field. The first results were apparently negative in the sense that no distinct objective symptoms arose, but as temperatures were not at first taken, it may be that reactions actually occurred. On account of the strong circumstantial evidence, the inoculation results, and the sterility of the tissues and fluids, however, the tick experiments were persisted with from season to reason.

Experiment 73 (Larvæ)—Lamb 299 (Sample experiment).—Between two and three thousand larvæ—*I. ricinus*—the progeny of females which had engorged on affected sheep in the field in May, were put on the scrotum of this lamb at the laboratory in September. On the sixth day the lamb was shewing uneasiness, constantly getting half up on its knees, and then lying down again. No particular attention was paid at first as the symptoms were not distinctive, and no temperatures were taken. On the seventh day the lamb was unexpectedly found dead. The lesions were typical of louping-ill.

Experiment 74—Lambs 292a and 289 (Larvæ and sub-inoculation with blood).—On the 19th September, 1916, two or three thousand larvæ of the same origin as those used on Lamb 299, were put on the scrotum of Lamb 292a.

On the fourth day the lamb was panting and uneasy, and seemed distinctly ill, lying for long periods, and only moving when made to get up, but showing nothing like complete paralysis. Smears from the blood shewed chromatin bodies; cultures from the blood were sterile.

Lamb 289 was inoculated into the muscles with 10 c.c. of blood drawn from lamb 292a when the temperature of the latter was 107°. A reaction occurred to 107°. This lamb was killed by bleeding for examination, and lesions referable to myelo-encephalitis were found on histological examination.

Experiment 82—Lambs 447 and 454.—On August 10th, one adult male and two female ticks were put to feed on lamb 447. The ticks had engorged as nymphs on a sheep affected with acute louping-ill in the field in May of the same year. They had moulted at the laboratory towards the end of July. Only one tick was observed to engorge on 447. The temperature chart (on screen) shews the reaction up to 107° which occurred. The usual lethargic symptoms appeared and were very evident on the twelfth day, but there was no definite paralysis. Chromatin bodies were found in the mononuclear cells in blood smears taken during the reaction. It appears from this experiment that the disease can also be carried by adult ticks which as nymphæ engorged on affected sheep, and incidentally it may be noted that in the case of adult ticks at least gross infestation is not a condition necessary to infection. Lamb 454 received subcutaneously 80 c.c. of blood taken from 447 when its temperature was 107.4°. The chart (on screen) shews a reaction up to 107.6°. The blood shewed chromatin bodies.

Having obtained these results with ticks and with blood of sheep infected by ticks it was indicated to inquire more methodically whether blood drawn in life from sheep while suffering from an attack of the disease was infective; a necessary factor to ticks becoming carriers is that an infecting agent is available in the blood of the vertebrate host.

Experiment 87 (Selected)—Ewe 317 and others.—This experiment, which included a considerable number of animals, is shewn in graph form on the screen.

Ewe 317 was inoculated with about 200 c.c. of blood, obtained during life, from an acute and ultimately fatal case of louping-ill in the field. The graph shews that the disease can be transmitted in series from susceptible animal to susceptible animal, provided the proper materials—blood, and gland juice—are used for inoculation, meaning that a living virus and not a toxin is involved. It also shews a negative reaction on the part of a heifer.

With regard to lamb 387—filtration experiment—it received 1,000 c.c. of blood which had been diluted for filtration, corresponding to about 50 c.c. of undiluted blood.

The rise of temperature to 106·4° occurred on the following day, and was almost

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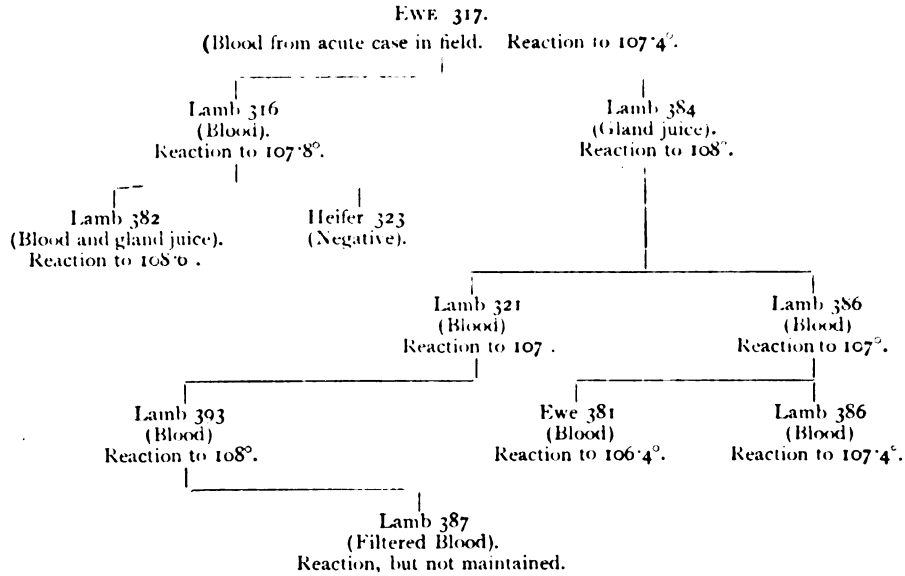
certainly due to the large amount of material which was injected into the hind leg, which caused lameness. After the second day it fell to normal.

GRAPH OF EXPERIMENT 87.



The negative result as regards material after filtration was obtained on several occasions with blood. More definite information, however, seemed desirable on the question of whether the virus was filterable or not. Accordingly a further experiment was designed, using an emulsion of œdematous lymph glands in which the virus seems to be specially concentrated.

Experiment 95—Ewe 461 and lamb 448.—Immediately after slaughter several glands, all more or less œdematous, were removed from a wether which was passing through a reaction after being inoculated with virulent blood. One gland from the lumbar region was slit open with a sterile knife, and a few drops of the thick juice were removed and transferred to a tube of sterile broth. The remainder of this gland and two others were cut up into small pieces, bruised in a mortar with a small amount of salt solution, and the resulting emulsion was passed through a Berkefeldt filter.

Ewe 461 received subcutaneously 5 c.c. of the unfiltered broth emulsion (reaction to 107·2°). Lamb 448 received subcutaneously 25 c.c. of a similar emulsion after filtration. (No reaction.) Charts shewn on screen.

Lastly, it remained to inquire whether ticks could be infected on the experimental sheep which were reacting to inoculation with blood or gland juice.

Experiment 96—Lamb 452. In the month of May, larvæ and nymphæ were put to engorge on a sheep while reacting to blood inoculation. The blood causing the reaction was drawn from another sheep which was reacting to blood obtained from a case of natural infection in the field. The ticks engorged, and moulted to nymphs and adults, as the case might be, by August, Some of these (nymphs and adults) were put to feed on Lamb 452 (Charts shewn on screen, reaction to 108°). Chromatin bodies were found in the mononuclear cells of the blood on the seventh day after tick infection, the temperature then being 108°.

* * * *

Many other questions in connection with this interesting disease have been inquired

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into; for example, it appears that after reacting to blood or lymph gland inoculation sheep are highly resistant to further inoculation. Sheep inoculated in the laboratory have also been exposed on infected pastures, and have come through a louping-ill season.

Again the question of whether ticks after infecting an animal become harmless has also been inquired into. The evidence so far obtained seems to shew that once having bitten on the sheep an infected tick loses its infectivity, but further work is being carried out on these questions.

Practical methods of prevention and eradication have also been studied, and it seems probable that the disease can be dealt with by either methods of prevention or eradication, which are practicable.

The result of the investigation might be summarised as follows:—

1. The blood and the juices of the œdematous glands from sheep affected with louping-ill in the field cause symptoms and lesions identical with those of louping-ill, the microscopic lesions being describable as a myelo-encephalitis as in natural cases; the blood and gland juices of the experimentally inoculated sheep are similarly virulent.

2. The larvæ from female ticks of infected sheep in the field, and adults which as nymphæ engorged on such sheep can convey the disease to other sheep; this means that infection passes through the egg. Similarly, ticks fed on sheep when reacting to inoculation with blood or gland juice may be infective to other sheep.

3. As the virus can be transmitted in series by inoculation, it follows that it is a living agent, and from the fact that it is transmitted by the tick it is almost certainly a protozoon parasite.

4. The causal agent is apparently not ultra-microscopic.

* * * *

It is the question of the causal agent to which I wish to draw your particular attention. If smears be made from the juice of the œdematous glands and stained by Giemsa's method, a particularly large number of what appear to be disintegrating mast cells are sometimes present. (Preparations were shewn under the microscope and on the screen). A number of the large and a few of the small mononuclear cells contain grouped or scattered bodies which stain a deep crimson-lake colour throughout. They vary from small dots under the oil immersion lens to objects of considerable size. They do not appear to have a highly differential structure, but in shape they may be round, oval, pyriform, or bacillary. They are mainly confined to the cell protoplasm, but in a few cases similar forms are to be seen in the nucleus against the lighter lilac tint of that body. The number of separate bodies in a single cell varies from one up to half a dozen or even more. Sometimes they are collected in nests giving the appearance of division having taken place, and fine strands of chromatin may be seen connecting two particles. In some of the cells the protoplasm may shew one or two clear or bluish spaces limited by a definite margin giving the appearance of a cyst, and inside are to be found a varying number of chromatin bodies of different sizes and shapes. By focussing carefully, a dark spot can sometimes be made out inside the bodies, but they stain so deeply that it is difficult to say whether a nucleus is present. The cell nucleus may be wholly or partially destroyed, the individual bodies being set free to enter or be taken up by other leucocytes, but for the purposes of interpretation it is important to note that many of the cells containing chromatin bodies are quite intact as regards the nucleus and cell protoplasm In blood smears made from a sheep suffering from acute louping-ill, or during a definite febrile reaction following upon inoculation with blood from a case of louping-ill, large mononuclear leucocytes containing similar oval and bacillary bodies are often particularly evident at the margin of the smears. As far as it is allowable to attribute a development to these bodies, or attempt to trace it by examination of fixed smears, it might be described as follows:—

A single oval body penetrates the leucocyte in which it increases in size and becomes more open like a rosette. It then appears to be become fragmented into smaller particles like the original, and divides up continuously. With a certain amount of reason one

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might construct a parasite of the hæmogregarine type (leucocyte variety) out of these findings, some of the bodies in the glands being regarded as trophozoites and merozoites, and the individual particles in the mononuclears of the blood stream as trophozoites and gametocytes. On the other hand, the chromatin particles, particularly those in the cells of the blood stream, might be nothing more than the debris of chromatin material which has been phagocytosed. It might also be suggested with a certain amount of reason that the chromatin particles result from chromatolysis of the nucleus, but in this connection it should be remembered that some of the cells containing the bodies are normal in every respect except for their presence. It is not claimed, however, that a final statement can be made on the evidence so far obtained that these chromatin bodies are parasitic in character, and the causal agent of louping-ill. Further investigation into their character is being carried out, but for purposes of discussion it seems allowable to summarise the evidence in favour of the parasitic view :—

(1) As has been shewn, the disease is tick-borne.

(2) The fluids and organs of sheep containing the bodies, and only such material, produce the disease by inoculation.

(3) The bodies stain somewhat after the manner of parasites.

(4) The causal agent of the disease is apparently not ultra-microscopic, since it is retained by filters as coarse as the Berkefeldt, and the chromatin bodies referred to are the only abnormal objects observable under the microscope in infective material.

Discussion.

Surg.-Capt. P. W. Bassett-Smith : The work done by Dr. Arkwright has been most thorough, and the results are most interesting ; but, as it is so late, there is not time to discuss the various controversial points. The way that he has met the continental criticisms, especially those of Brumpt, are valuable, the infected pediculi used by the latter being derived from German prisoners, which, however, did not pass on trench fever, as might have been expected.

With relation to trench fever, Dr. A. C. Coles has sent a communication of work done, and has shewn me spirochætal forms in films of blood taken from successfully experimentally inoculated cases which were sent to him by Major Byam, R.A.M.C., from the Hampstead Hospital, by permission of Sir David Bruce.

The spirochætal bodies were found after prolonged search in the blood films of two out of six cases, in the first paroxysm of the fever. Photomicrographs were exhibited.

The spirochætes all stain a delicate blue with Giemsa, and are faintly granular, and vary considerably in form. Dr. Coles states that they cannot be regarded merely as artefacts, but the fact that they were detected only in definite cases of artificially induced trench fever, and then only during the first attack, is at least suggestive. These findings, in conjunction with the work done at the Pasteur Institute, should encourage still further research as to the spirochætal origin of trench fever.

Professor W. J. Simpson : It is very difficult to collect one's thoughts and do justice to the papers that have been read without having had an opportunity of seeing and studying them beforehand. Indeed it would be an advantage if such a plan could be adopted. The two papers represent much the same stage of research in their respective spheres. Sir Stewart Stockman shews the presence of chromatin bodies in great numbers in louping-ill, and establishes the fact that the infection is continued from one generation to another, and that these infected ticks produce the disease. The veterinary department have the advantage over the medical department in being able to conduct their experiments to a final conclusion. Sir Stewart Stockman hopes to be able to isolate these chromatin bodies from the juices and any unseen organisms that may possibly be present in these juices, and we shall look forward to his further contribution on the subject.

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Dr. ARKWRIGHT's description of the discovery of the Rickettsia bodies in trench fever and in the louse, and the rôle that the louse plays in trench fever, demonstrate that this investigation has reached a very advanced stage, and it is to be hoped that the next will be the isolation and cultivation of these bodies, and the production of the disease with the cultures. There are many pitfalls, however, in such investigations, and one has to be particularly cautious. I have fallen into the pitfalls myself. I have been interested in the cultivation of vaccine nearly all my professional life, and when in India succeeded in cultivating from a buffalo with rinderpest, but which, in this instance, I consider, was a form of smallpox, a minute diplobacillus, which produced in calves a vaccine vesicle, the lymph of which produced vaccine vesicles in other calves and children, who were thereby rendered immune against subsequent vaccination with ordinary vaccine lymph. I lost the strain of culture in the rains. I have never been satisfied in my mind whether the minute diplobacillus was the actual causal agent, or whether it was merely the visible companion of a more minute and invisible organism in symbiotic relation with it. Such a possibility has always to be borne in mind in these investigations. I would ask the Fellows to accord a hearty vote of thanks to the authors of the papers read this evening.

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HAS EMETINE ANY INFLUENCE ON THE SCHISTOSOMES?

BY

C. BONNE, *Paramaribo, Dutch Guiana.*

In March, 1916, I treated an East Indian immigrant, who suffered from amœbic dysentery, with emetine. The patient received each day a subcutaneous injection of one grain of emetine hydrochloride on five successive days, with the usual good effect on the dysenteric symptoms. The stools of this man had been containing also ova of *Schistosomum mansoni*, and I was rather astonished when I could not find these eight days afterwards. I repeated the examination on sixteen following days always with the same negative result.

This made me try a series of five injections of one grain of emetine with other patients shewing the eggs in the fæces, the results of which, together with those of the first patient, are given in the table below. Each time at least three slides and, as a rule, 4–12 were examined either by myself or by trained assistants when the eggs could not be found. In several of the cases I put a premium on the finding of the eggs, which, as the table shews, I often lost in the end.

The results can be summarised as follows:—

1. As a rule the eggs disappear from the stools after about a week. In a few cases they do not disappear, but they are found less frequently (12, 16, 18), or even this is not obvious (19), but then they may still disappear after a second course of injections (16, 18).

2. After about two months, sometimes sooner, already the eggs are found again, but can be made to disappear by a second course of injections.

Point 1 can be explained as follows: It takes about a week for the eggs to work themselves out of the tissues into the lumen of the rectum, and no new eggs are laid in the wall of the rectum by the worms for some reason, *e.g.*, they may be dead or have moved away from the veins there to the larger veins of the portal system.

Point 2 can be explained by the worms returning to the rectum wall, either the old ones or other worms, *e.g.*, younger worms, which had not yet left the portal vein when the injections were given. The reappearance of the eggs is too common to be explained by reinfection.

Schistosomiasis is not a severe disease in Dutch Guiana, at least not in the very large majority of the cases. Nearly all the patients of the Table shewed no intestinal symptoms at all, and the eggs were only found in the routine examination of the fæces. When they had intestinal symptoms, dysentery like symptoms, for instance, I very often found amœbæ or dysentery bacilli by which they could be explained. I believe that the presence of the schistosomes, probably the passage of the eggs through the mucosa, predisposes to infections with dysenterial organisms. On the other hand, I met with cases where no such organisms could be found and still there was passage of mucus and blood and abdominal pain. The emetine has a beneficial and often a very striking effect on these symptoms. This is readily understood when amœbæ are present, but in the other cases there must be some other action on the diseased mucosa. It is always possible, however, that a small number of amœbæ were at work but not discovered. I

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think one must not too hastily ascribe all sorts of abdominal troubles to the schistosomes, when eggs of *Schistosomum mansoni* are found. There is no doubt, of course, that in some cases the worms are able to cause serious damage. In Dutch Guiana, however, this is rare. I never met with a case of death due to schistosomiasis, although in the course of three years more than two hundred cases have entered the hospital in Paramaribo, nearly all of them for other diseases. Patient number 4 of the Table had a definite polyposis recti, and a few of the tumours removed by operation contained a great number of the eggs.

It was necessary of course to control the patients during a long time, and it was very difficult to get the fæces of these people for examination when they had left the hospital. This was one of the reasons why I did not continue these controls. Then I did not like the explanation of the disappearance and reappearance of the eggs, because it was so hypothetical, and I saw no chance to prove or to disprove it. It was desirable to know whether the emetine killed the worms in the rectum wall or not, and this could only be proved by a post-mortem shortly after a course of injections. I never had such a chance. It would be worth while to know if one could be freed permanently of the eggs in the fæces by an intermittent treatment with emetine. But as the persons that I saw harbouring the worms had not suffered from them for a long time, they declined to receive these repeated injections. It is even possible that there was no influence on the schistosomes at all, and merely on the mucosa through which the eggs have to pass. The results obtained, however, can hardly be ascribed to mere chance.

DIAMANTIS had some results with emetine in bilharzial hæmaturia in Egypt (*Trop. Dis. Bull.*, Vol. XI. No. 2). MARTIN MEYER noticed improvement in a patient suffering from urinary and intestinal bilharziasis, and VAN HOOF in a patient with rectal bilharziasis (*Trop. Dis. Bull.*, Vol. XII., No. 3). It was only after I had read the results of these authors that I thought it worth while to publish my own experiences. They leave no doubt that emetine has an influence on the symptoms in rectal schistosomiasis due to *Schistosomum mansoni*, but I cannot say whether the drug is able to kill off the worms.

On the following page is a Table shewing the influence of emetine on the presence of eggs of *Schistosomum mansoni* in the stools :—

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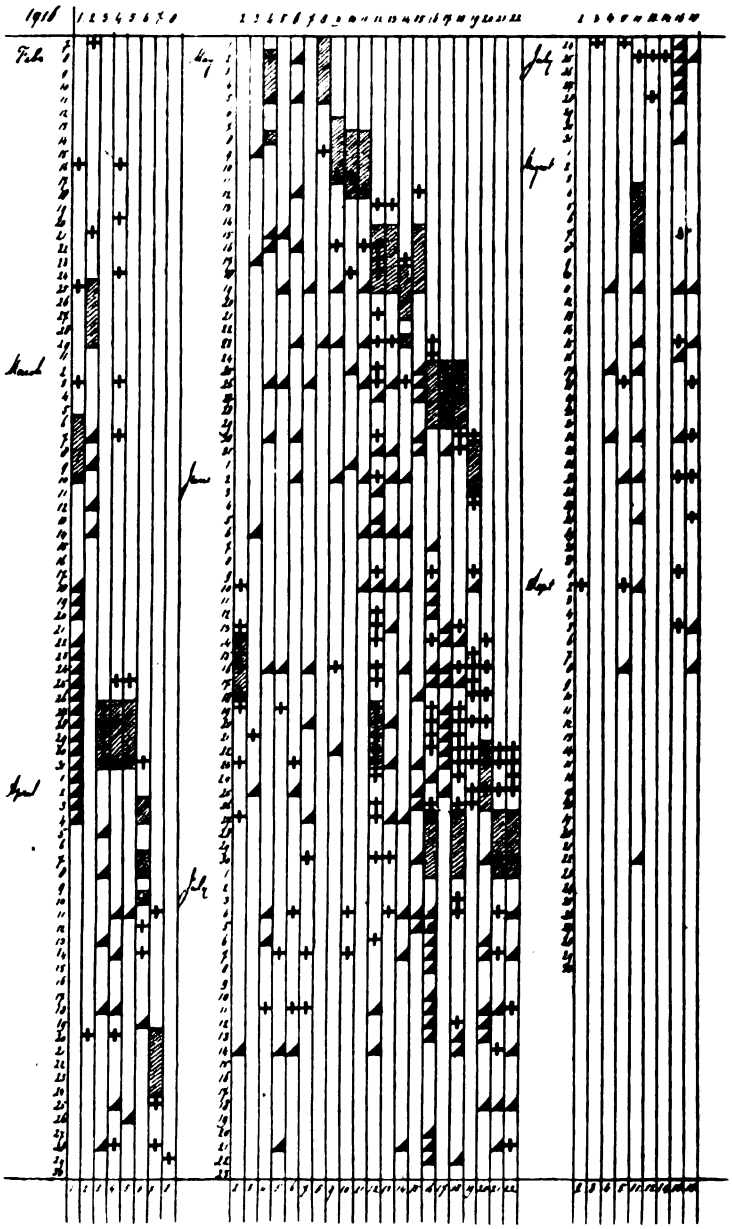

TABLE SHEWING THE INFLUENCE OF EMETINE ON THE PRESENCE OF EGGS OF SCHISTOSOMUM MANSONI IN THE STOOLS.

+ means eggs found. ◢ means eggs not found. ≋ period of emetine injections.

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RAPHAËL BLANCHARD.

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Obituary.

RAPHAËL ANATOLE EMILE BLANCHARD

(1857—1919),

HONORARY FELLOW OF THE SOCIETY.

RAPHAËL BLANCHARD was born February 28th, 1857, at Sainte-Christophe (Indre-et-Loire), in France.

His father, RENÉ BLANCHARD, who died at the early age of twenty-six, was a poet and author. The driving force which distinguished RAPHAËL from the start is, perhaps, best exemplified by reference to his grand-uncle, JEAN PIERRE BLANCHARD (1753-1809), the aeronaut, who first devised the parachute for use in aeronautics in 1785. JEAN tested the apparatus on a dog enclosed in a basket, and, in 1793, himself descended from a balloon by this means, although he broke his leg in the proceeding. He was the first aeronaut to make a voyage across the British Channel, flying from Dover to France in a balloon (7th January, 1785).

In 1874 RAPHAËL BLANCHARD went to Paris to take up the study of medicine and natural science, and in 1875 he became assistant to CHARLES ROBIN. In 1876, with others, he founded the Societé Zoologique de France, of which he became the Secretary-General and moving spirit for twenty years. The year 1877-78 was spent by him in Austria and Germany, after which he returned to Paris and became assistant to PAUL BERT (1878-83). In 1878 he taught natural history at the Lycées Saint-Louis and Louis-le-Grand, and in 1884 gave instruction at the École d'Anthropologie. In 1883 he became Associate Professor at the Paris Faculty of Medicine, and started the first courses of Parasitology that were delivered in France. In 1889, with MILNE EDWARDS, he organised the International Congresses of Zoology, in which he took an active part, especially in establishing the Rules of Zoological Nomenclature that have since been so widely adopted. It was owing to his energetic efforts in this direction that he was made President of the International Commission on Zoological Nomenclature in 1898. In 1894 he became member of the Academy of Medicine, and in 1897 was elected full Professor of Medical Zoology, the title of this chair being changed nine years later to that of Parasitology at his own request. With others, in 1902, he founded the Societé Française d'Histoire Médicale, of which he was president during the first three years. In the same year was inaugurated the Institut de Médecine Coloniale, with its hospital, whose foundation was brought about by BLANCHARD, with the aid of funds supplied by the Association des Dames Françaises. He was Annual Secretary of the Academy of Medicine from 1912 until his death. Throughout the war he gave devoted service to his country in promoting more particularly the work of the Association des Dames Françaises.

BLANCHARD's published work ranged over a wide field. A full account of it will be found in a series of brochures, entitled *Notices sur les Titres et Travaux Scientifiques de M. le Dr. Raphael Blanchard*, the first of which was published at Lille in 1890, whilst two supplements appeared in Paris in 1893 and 1908; a third and final supplement is now being prepared for press. The complete lists of publications contain no less than 603 titles of books, brochures, original papers, short notes, articles that appeared in encyclopedias, circulars, etc, some of which were translated into various languages from the French. A good list of his publications, covering nine pages, will also be found in Stiles and Hassall's *Index Catalogue of Medical and Veterinary Zoology* (Washington, 1902).

His publications relating to Parasitology are the most important; they deal with parasitic worms, leeches, protozoa, moulds, myiasis, injurious or disease transmitting

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insects, mosquitoes, acari, pseudo-parasites, myriapods, and touch on various tropical diseases. He published numerous papers on zoological nomenclature, general zoology; on comparative anatomy, histology, physiology, pathology and teratology. He likewise ventured into the domain of anthropology, wrote on medical history, education, and on such miscellaneous subjects as numismatics (he possessed an extensive collection), book plates, and sundials!

Of the larger works may be mentioned the *Elements de Zoologie* (written with PAUL BERT, 1885), his *Traité de Zoologie Médicale*, in two volumes (1885–1890); the *Histoire naturelle et médicale des moustiques* (1905), the *Epigraphie médicale*, of which four fasciculi have appeared since 1907, this work dealing with epitaphs, etc., having medical interest. Finally must be mentioned the *Archives de Parasitologie*, a quarterly journal founded by him in 1898, and that he edited and owned.

The foregoing summary indicates sufficiently the remarkable character of BLANCHARD's activities. He was an indefatigable research worker, a good organiser, a highly gifted and versatile man. All who knew him felt that he was the embodiment of energy and enterprise. He had a considerable command of languages, and was an eloquent speaker, qualities which rendered him eminently fitted to attend international congresses. He participated frequently at these meetings, and his well-known shining figure will be greatly missed at future congresses of medicine and of zoology. He had many warm friends; that he should have had some enemies is natural, for such is the fate of most leaders of men.

RAPHAËL BLANCHARD died on February 7th, 1919, at the age of 62 years, with a suddenness that came as a great shock to all his friends. He had had what he supposed to be merely a severe cold, and which gave those about him little concern. He was arranging to begin his lectures on February 10th, when suddenly, without warning, he had an attack of syncope and passed away.

It seems fitting to conclude this obituary notice with a passage translated by the writer from a short biography written by LINOSSIER (*Paris Médical*, 15 fév., 1919) a few days after his friend BLANCHARD's death: –

"BLANCHARD led a brilliant existence. Life smiled upon him. He knew little of its bitterness, and he had a lively appreciation of its joys. Honours were showered upon him, and he received them willingly. He had arrived at the summit of his ambitions. Then death, seeing him happy, and fearing for him the inseparable afflictions of the finest old age—sadness and melancholy—desired to shield him from them: considerately and gently she spared him the agony of her approach—suddenly she struck him down by a touch of her wing, and bore him away tenderly without giving him pain. . . ."

20th April, 1919. G. H. F. NUTTALL.

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TRANSACTIONS

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VOL. XIII.

1919-20.

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TRANSACTIONS

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SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

MAY 16TH, 1919.

VOLUME XIII. . No. 1.

Proceedings of a Meeting of the Society held on Friday, May 16th, 1919, at 5.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Surgeon-General Sir DAVID BRUCE, K.C.B., F.R.S. (*President*), in the Chair.

FLY-SPRAYS.

BY PROFESSOR H. MAXWELL LEFROY.

Imperial College of Science, S. Kensington.

In July, 1915, a bottle containing a few ounces of a spirituous fluid was sent to me by the Army Medical Department, with the information that it was claimed to have curious properties with regard to fly destruction. It had been received from Mr. O'SULLIVAN BEARE, who had lately arrived in England from Brazil, and who learnt from the *Daily Mail* that flies were a factor in the transmission of disease in the war areas. Mr. O'SULLIVAN BEARE told me that in Brazil it was in general use, especially by travellers, who sprayed this liquid in the rest-houses where they stayed, with the result that flies, mosquitoes, and all other insects were exterminated and the traveller spent a peaceful night. He wished to be sent to Brazil to obtain immense quantities for the army; the only information he gave as to the nature of the liquid was that it consisted of an alcoholic tincture of a plant common in Brazil.

I had to test this liquid, and I was not then aware of any similar liquid against which to test it; but I decided to use a high-flash mineral oil, to which was added a small amount of essential oil to cover the smell. An equal number of flies was released in two rooms; an equal quantity of the tincture and of the oil was sprayed in each room; the results were that with the tincture practically all the flies came down in a peculiar condition of helplessness, while with the oil nearly all the flies came down either helpless or dead. If anything, the oil spray was the better of the two, and report was made accordingly. Mr. O'SULLIVAN BEARE furnished no further information as to the fluid, and, as no more was available, nothing more could be done then. But as the method of spraying small amounts of mineral oil did, to some extent, destroy flies, a large supply of

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the oil was sent to the Mediterranean area, and this constitutes the first use known to me of the fly-spray method in the war.

This oil was an ordinary paraffin gas oil or light fuel oil, with a boiling point about 300°C. and a specific gravity of about 0·820; it is the grade of oil heavier than the burning oils, lighter than the lubricating oils, and to it was added 5 per cent. of citronella oil.

From August to October, 1915, the question of systematically working out tests of fly-sprays was gone into, and it was found that an extract of pyrethrum powder in spirit was an active fly-spray, even when much diluted.

The standard extract used eventually was one pound of pyrethrum powder to half a gallon of spirit, this being carefully percolated or slowly macerated, cold.

In Appendix I., a table is given of tests of a few extracts used with the results; it would be useless to tabulate all of these, and in Appendix II. is a list of the substances tested.

The ultimate outcome was this: a standard spirit tincture alone was good, if diluted in water up to about ten times; of other things tried, safrol was about the next best, working well in emulsion up to about 2 per cent., but the combination of pyrethrum extract with safrol was the best mixture. The final formula was:—Pyrethrum, 2lbs., spirit, 1 gall., safrol, 1 gall., soap, about 1oz., or enough to make the mixture emulsify in water. Dilute at 1 to 30 and spray.

The work that was done was closely followed by the Army Medical Department, and particularly by Major LELEAN, Professor of Hygiene at the Army Medical College. The fly-spray as described was adopted by the Army Medical Department, and first used in Salonica.

In testing fly-sprays, two methods were used: the first and usual one was to place a number of flies in a wire balloon trap as ordinarily used, and to spray into it 1 c.c. of the liquid; the flies were then observed at intervals and results noted. The second was to release a large number of flies in a small greenhouse and to spray 100 c.c. of the diluted liquid and to note the results.

In November, 1915, I was sent to India, and while there was sent to Mesopotamia to deal with flies. Before going, I arranged in Bombay for the making of the fly-spray, and also obtained a large quantity of a light lubricating oil (velocity spindle oil), to which was added citronella. The pyrethrum-safrol spray failed altogether in Mesopotamia; the oil spray was satisfactory up to a certain point. When I returned in December, 1916, to the Imperial College, experiments were started to ascertain if the ordinary fly-spray could be made suitable to hot climates, and I was assisted very ably in this by Mr. L. LLOYD. We rigged up the small greenhouse so that it could be heated to 115°F., and in that temperature we tested fly-sprays. We found that the addition of a small amount of castor oil, which is the only vegetable oil soluble in alcohol, made the normal pyrethrum-safrol spray effective and the final formula contained from ½ to 2 per cent. of castor oil. This was communicated to the Army Medical Department, and, I understand, was adopted.

During 1916, another fly-spray was used in the army, which was actually only the original oil-spray with peppermint oil instead of citronella. An alcoholic tincture, which the makers claim to be made to the formula of Mr. O'SULLIVAN BEARE, was also in use. I understand that this latter was used with success in Egypt, but, as it was used neat, the cost was high. The only other fly-spray known to me is an American one, which was sent to the Army Medical Department. It was effective but costly, a gallon selling in America for four dollars. It was stated by the makers to be an oil distilled from the needles of a pine.

The action of fly-sprays is not understood. It is known that pyrethrum contains an oil, and a complex organic substance which has been called pyrethro-toxic acid, the latter having an action on the nervous system of insects which is far more intense than that of any other known substance. In the fly-spray there is actually 1 part of pyrethrum in 100 parts by weight of water That 1 part of pyrethrum contains only a very small amount of the complex organic substance, far less than 1 per cent. But even if 1 per cent. were present, it would be working out at 1 in 10,000. Nicotine is used at 1 to 1,000, and that is one of the substances reckoned to be very

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toxic to insects. Safrol is chemically the allyl. 3. 4. pyrocatechol-methylene-ether ($CH_2.O.O.C_6H_3.CH_2.CH.CH_2$), allied to anisol and phenetol, both ethers in which a benzene and an ethyl or methyl group are joined by the oxygen atom, and all three are excessively toxic to insects. How these act on the insect is not known, but the probable action is that they combine extremely rapidly with compounds in the nervous system, and prevent further combination with oxygen, and so produce paralysis or inhibition of further chemical and physiological action.

Dr. BARGER, who investigated the chemical nature of the active substance in pyrethrum powder, isolated the active substance from petrol extraction of pyrethrum, but I am not aware of anything published by him. We tried a series of solvents to secure the maximum extraction from the powder, but though petrol is said by Dr. BARGER to be the best, spirit was used throughout, as petrol presented great difficulties in use.

Fly-sprays are useful under conditions where other methods of fly destruction are inapplicable. For rapidly clearing buildings, they are excellent. For the open they are useless. In the tropics particularly, where flies and mosquitoes are a burden, fly-sprays should have a great use.

The sprayers used have been the common type of tin sprayers, in which air is pumped through a narrow pipe, which opens across the opening of a pipe leading from a liquid container; as the air blows past it sucks up the liquid and atomises it. Used properly, a room or building can be very quickly and easily cleared of flies.

Since this work was completed, a great mass of further work has been done on the relationships of chemical structure of the molecule and toxic action on insects; and I have had occasion to use other substances than those named as fly-sprays. For this purpose, flies have been placed in wire balloons in a long glass box, 10 feet by 1 ft. by 1 ft., and a cubic centimetre of the liquid atomised in at one end. It was possible to get a measure of the toxicity by seeing whether the effect extended along to the whole 10 feet or only to part, or yet again, whether there was no effect. In Appendix III. the results are given of the compounds so tested. They are of interest and they shew that safrol is by no means the best substance and that a large field for research lies open. I am led to think that possibly quite new substances will be used as fly-sprays when this subject is further investigated, and I hope that ultimately we shall get substances as efficient as pyrethrum, which can be made pure, and in which we shall not have to depend on the small amount present in the plant. If so, then the use of fly-sprays should become more general even in England. In my own case, I spray a small amount of liquid in my kitchen in the hot summer weather at night. I can kill all the flies, and by doing this every few days I can check the plague of flies that otherwise abounds from July to September.

Particularly I commend the method for the tropics, where not only flies but mosquitoes, plant bugs, beetles, etc., are such a plague as soon as the lamps are lit. Pyrethrum and spirit can both be obtained, and if safrol is difficult, at least a spirit extract of pyrethrum can be made and sprayed with success. Probably when wider publicity comes, chemists in the tropics will make up and sell fly-sprays as well as supplying tin sprayers, and medical and sanitary officers will be able to see that the ingredients are available.

APPENDIX I.

I.—MUSCA IN BALLOONS.

A.

	No. of Strokes. (20 = 1 c.c.)				Results.
Pyr. Ext.	20	1 hr. *l*	5 hrs. *l*	22 hrs. 2 *w* 40 *l*	... *l*
,, + H_2O 5% ...	20	1 ,, *l*	5 ,, 2 *w l*	22 ,, 17 *w* 14 *l*	... *w l*
,, 50% + alc. (90%) ...	20	1 ,, *l*	5 ,, 5 *w l*	22 ,, 22 *w* 13 *l*	*w l*
,, 75% + H_2O 25% ...	20	—	5 ,, 7 *w l*	22 ,, 11 *w* 1 *l*	... *w*
,, 10% + H_2O 90% ...	20	1 ,, 9 *w* 5 *l*	—	22 ,, 14 *w*	... *w*
,, 5% + H_2O 95% ...	20	—	—	22 ,, 10 *w*	,, *w*

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	No. of Strokes (20 = 1 c.c.)					Results
Oil	20	—	—	—	22 ,, 10 *d*	*d*
,, + cinnamic ald. 1°/₀	20	—	—	—	22 ,, 16 *d*	*d*
,, + lavender oil 5°/₀	20	—	—	—	22 ,, 15 *d*	*d*
,, + pine oil 5°/₀	20	1 hr. 4*t* 16*d*		—	22 ,, 4 *w* 16 *d*	*w d*
,, + iodoform 1°/₀	20	—	—	—	22 ,, 18 *d*	*d*
Miscible oil, light	20	—	—	—	22 ,, 7 *d* 10 *w*	*w d*
Misc. heavy oil 20°/₀, cinn. ald. 4°/₀, H_2O 76°/₀	20	—	—	—	22 ,, 7 *d*	*d*
Misc. heavy oil 10°/₀, amyl alc. 10°/₀, H_2O 80°/₀	20	—	—	—	22 ,, 2 *t* 1 *d*	*t d*
Misc. Oil + 5°/₀, fusel oil 20°/₀	20	—	—	—	22 ,, 11 *w* 4 *d*	*w d*
Amyl alcohol 50°/₀, H_2O 50°/₀	—	—	—	—	22 ,, 6 *w*	*w*
,, 10°/₀, solv. soap 10°/₀, aq. 80°/₀	—	—	—	—	22 ,, 10 *w*	*w*

w, Well. *d*, Dead. *t*, Alive, but unable to walk or fly.

B.

	24 hours.
10°/₀ miscible heavy oil, 10°/₀ of 1 in 25 petrol pyr. p.	1 *w* 8 *d*
Petrol ext. of *derris*	6 *w* 1 *t* 1 *d*
1 gr. pyr. p. in 25 c.c. oil in 225 c.c. aq.	1 *t* 4 *d*
2 grs. ,, 50 c.c. ,, 450 c.c. ,,	6 *d*
1 c.c. solv. soap, 1 c.c. oil, 8 c.c. aq.	4 *w*
1 c.c. solv. soap, 1 c.c. of 5 gr. pyr. p. in 25 c.c. oil, 8 c.c. aq.	1 *w* 6 *d*
1 gr. Bidwell 2, 10 c.c. petroleum	5 *w* 1 *d*
1 gr. ,, 3, 10 c.c. ,,	4 *w*

D.

1 c.c. solv. soap, 1 terpineol, 8 aq.	18 *d* 11 *w*
1 c.c. ,, 1 petroleum 8 aq.	0 *d*
1 c.c. ,, 1 petroleum ext., pyr. p. (1/10), 8 aq.	3 *d*
1 c.c. ,, 1 ,, pyr. p., 8 aq.	5 *w*
1 c.c. ,, 1 ,, pyr. p., 8 aq.	10 *w*
1 c.c. terpineol, 9 c.c. alcohol (90°/₀)	4 *w* 3 *d*
5 c.c. (terpineol 1 alcohol (90°/₀) 9), 5 (1 solv. soap, 1 petroleum, 8 aq.)	6 *w* 2 *d*
1 c.c. terpineol, 1 ext. Bidwell 3, 8 aq.	3 *w* 6 *d*
1 c.c. solv. soap, 1 ,, 2, 8 aq.	4 *w*
1 c.c. ,, 1 terpineol ext. pyr. powder, 20 aq.	4 *w* 1 *d*
Pyr. ext. I. 2, turpentine 2, aq. 16.	all down, ⅕ recover
,, ,, safrol, ,,	all down, die
,, ,, turpentine, ,,	,, ,, ⅕ recover
Safrol 2, soft soap sol. 2, ,, 16.	,, ,, alive but torpid
,, 1, ,, ,, 1, ,, 8.	,, ,,
,, 1, ,, ,, 1, ,, 38,	,, ,,
,, 1, pyr. ext. 1, ,, 20,	,, ,,
,, 1, ,, 1, ,, 40,	,, ,,
,, 1, ,, 1, ,, 100,	,, ,,
,, 1, soap sol., 1, ,, 100,	,, ,,

	1 hour.	24 hours.	48 hours. Dead.	Well.
Cinol 1 in 10	down	⅓ up	120	19
Pyr. ext. + soap 1 in 10	,,	½ up	35	13
,, + ,, + cinn. ald 1 in 10	,,	⅓ up	100	38
Hellebore ext. 1 in 10	many down	all up	12	40
Acetic pyr. ext. 1 in 10	,,	⅔ up	all well	
Pyr. ext. + safrol 1 in 10	all down	all down	60	0
,, + ,, 1 in 20	,,	,,	90	0
,, + borax	down	$\frac{6}{50}$ up	104	12
Safrol and soap 1 in 40 each	,,	down	96	0
Misc. oil (light) + pyr. ext. 10°/₀ each	,,	⅓ up	62	30
Glycerine pyr. ext. in alc. 1 in 5	,,	⅖ up	33	25
Turpentine ext. 1, solv. soap 1, water 8	,,	½ up	25	11

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APPENDIX I.—*Cont.*

	IMMEDIATE.	24 HOURS. Dead.	Well.
Safrol 1 in 100	down ...	20	21
,, 1 in 60	,, ...	60	14
,, 1 in 50	,, ...	22	18
Safrol + pyr. ext. 1 in 100	,, ...	68	0
,, + ,, 1 in 60	,, ...	37	0
,, + ,, 1 in 50	,, ..	70	0

APPENDIX II.

Pyrethrum. — Powdered dried flower-heads of *Pyrethrum*, either Dalmatian or Japanese
Cinnamic aldehyde
Lavender oil
Pine oil
Iodoform
Miscible oil (light, heavy, or with fusel oil)
Amyl alcohol
Derris (MacDougall) powdered *derris* root
Mineral oil (A.D.O.)
Bidwell's powder, unknown plant
Terpineol
Acetone
Trichlorethylene

Solvent Soap = sulphonated castor oil
Petrol
Phenetol
Turpentine
Carbon bisulphide
Ethyl acetate
Formalin
Safrol
Anilin
Camphor
,, oil
Hellebore
Acetic Acid
Glycerine
Geraniol

Borax
Stavesacre
Piperazine
Brombenzene
Dichlorbenzene
Ethylthiocyanide
Oenanthol
Pseudocumol
Olive oil
Terpinhydrate
Picrotoxin
Canada balsam
Capsicum
Copaiba balsam
Balsam of Peru
,, of ginge

APPENDIX III.

DETERRENTS.

Paraffin	0	
Petroleum acid tar	0	
Acid formic	0	Excitement.
Chloroform	0	
Tetrachlorethane	2	
Bromoform	2	
Acetone oil, yellow	0	Escape.
,, brown	3	Down, die.
Acetamide	0	
Anethol	3	Down.
Aniseed oil	3	
,, terpene	3	Down, torpid in corners.
Phenetol	1	
Safrol	3	
Eugenol	1	Excitement.
Ethyl acetate	2	Excitement.
,, oxalate	1	Excitement.
Phorone	2	
Toluene	2	
Naptha crude	0	Excitement.
Soluble creosote	0	
Cresol, 95 per cent.	3	Mad, down.
Green tar oil	1·2	Effect not constant.
Light creosote, N.C.C.	2	
Benzyl chloride	2	
Nitro benzene	2	Drunk.
Aniline	2	Down.
Crude aniline oil	3	Down, no recovery.
Methyl salicylate	2	Excitement
Terebene opt.	0	
Citronella resin	2	Torpid, refuse food.
Light wood oil (Greenwood)	3	Down.
,, ,, (Cartvale)	3	
Wood creosote	2	
Dipentene	2	
Copal oil...	3	

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APPENDIX III.—*Cont.*

Varnish fumes	3	
American fly-spray	3	Down.
Crude resin oil (Dussek)	2	Not down.
Refined ,, ,,	2	
Copaiba balsam	1	
Clayton's fluid	3	Down well.
Pyridine	2	Drunk.
,, crude	2	,,
Heavy tar bases	2	,,
Pyridine (Hardman)	2	Down.
Dippel oil		Drunk.
Nicotine, 98 per cent.	3	Mad, down.
Aniseed	2	
Carraway (Carvone)	2·1	
Origanum (Carvacrol)	2	
Cassia, 80 per cent. Cinn. aldehyde	2	
Cinnamon bark	2	
Eucalyptus globulus (cineol)	2	
Lavender (Esters + cineol)	1	
Verbena-citral	2	
Peppermint (menthol, menthane)	2	
Sassafras (safrol)	3	
Br. camphor (safrol)	3·2	
Thyme-thymol	2	
Thymene, residue of thyme	3	Down.
Pine oil	2	
Cajeput	1·2	
Essential oil resins	2	
Eucalyptol	2	Not dead.

The figures are the index of deterrent action :—

0 = no action. 1 = moderate. 2 = definite. 3 = extreme.

All substances with 3 are likely to be useful in fly-sprays.

Discussion.

Major E. E. Austen: May I be allowed to congratulate Professor Maxwell Lefroy on the painstaking investigations of which he has just given us an account? It is very evident that an enormous amount of work has been done, and that the results of such an extensive series of experiments are bound to be extremely valuable. I am not quite clear whether, in his researches, Professor Lefroy was envisaging the needs of troops in the field or of civil populations in ordinary times of peace. The two cases are of course totally different. One's thoughts are still almost inevitably coloured by the events of the last four and a half years, and it is as yet a little difficult to dissociate oneself from war, and think solely of a civil community. While I can quite imagine an effective fly-spray being of the greatest use in a town or village, or even under conditions which obtain at a military base in time of war, I am bound to say that such experiments as I have made in Mudros, Sinai and Palestine corroborated what Professor Lefroy told us, and pointed to the conclusion that the spraying method, as a means of fly-control, is practically useless in the field, at any rate in warm climates. The fluid used was Hepple's army spray, which I believe is identical with the alcoholic extract of pyrethrum, plus safrol, spoken of by Professor Lefroy. Apart from the difficulty—by no means a negligible one when lines of communication are long—of getting sprayers and fluid to spots at which they are wanted, and in addition to the apparent lack of killing power in the fluid itself, it was found that, in the field, flies in hot weather do not afford good "targets" for the sprayer. That is to say, until the nights begin to get cool, the insects do not usually enter tents and bivouac shelters in numbers sufficient to make the use of a sprayer worth while, even assuming the efficiency of the fluid used. So far at any rate as the E.E.F. was concerned, results shewed that infinitely the best method

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of fly-control was the prevention of fly-breeding and fly-contamination, ensured by a fly-proof latrine-system, coupled with the prompt incineration of human excreta and camp refuse, and properly controlled spreading of horse-line manure. Wherever possible, these methods were supplemented by the use of poisoned bait (arsenite of soda, or formalin). It must be admitted, however, that poisoned bait of proved efficacy in one locality may be a total failure in another, or under a different set of conditions. Thus arsenite of soda, though very effective at Rouen, in June, 1915, was completely useless at Mudros in October of the same year, even with the addition of double the normal amount of sugar.

There is just one question which I should like to put to Professor LEFROY, and in asking it I need scarcely say that I am actuated solely by a desire for information. At Rouen, in June, 1915, pyrethrum powder, diffused through the air in quantity sufficient to produce a colourable though faint imitation of a London fog, was found absolutely effective in destroying every single fly inside a closed Aylwin hut. The method was tried night after night with complete success. This being so, why is it considered necessary to go to the trouble of making an extract of the powder, for use in a sprayer?

Lieut.-Col. A. BALFOUR: I am anxious to obtain some information from Professor LEFROY because I am not clear if the castor oil of which he spoke as rendering sprays more effective in the tropics is added to the light fuel oil-spray or to the pyrethrum-safrol spray. I believe it is to the latter, but Professor LEFROY did not seem to me quite clear upon this point. I have recently had to write something about preventive measures for flies, and have come across accounts of "Cinol" fly spray and of the "Osbeer" fly-spray, which is also called flybane. If Professor LEFROY can enlighten me as regards their composition I should be grateful. I may say that I have seen gallons of fly-spray wasted in several parts of the tropical world, but I have also seen it usefully employed, as, for example, in the dug-out of a certain General at Cape Helles, where, shortly after the arrival of Army fly-spray on the Gallipoli peninsula, we spent a profitable evening killing flies by means of one of the tin Hepple-sprays which Professor LEFROY mentioned as suitable for indoor use.

I quite agree with Professor LEFROY and Major AUSTEN that fly-sprays are of little use in the open, but I have seen them do some good. I remember seeing a considerable number of flies killed by what was probably the light fuel oil-spray in an open courtyard at Amara. This, however, happened in November, when the conditions were comparatively cool, at least for Mesopotamia.

I should like to ask Professor LEFROY whether the efficiency of this addition of castor oil has been actually tried in hot climates, or whether it has merely been tested experimentally in this country.

Mr. A. W. G. POMEROY: In reference to the point brought forward by Major AUSTEN, that in the field under war conditions in the tropics, fly-sprays were generally found to be an impracticable method of control, I beg to agree entirely.

In Dar-es-Salaam, East Africa, where I was working on fly control for some time, the greatest centres of attraction for flies were such places as supply dumps, canteen stores, butcheries and remount depôts.

Spraying in the first two instances was abandoned owing to the damage real or fancied which might occur to the stores from the chemicals used, especially on stores such as dates and potatoes, of which there were large quantities.

In the other two main centres there was never found a sufficient percentage of flies congregated in one place to form a suitable target, so that they would not be decreased by this method to the same degree as by the more simple one of destruction in the early stages.

The activity of flies in the tropics I have found to be very different from their usual activity in the temperate climates.

Many times I have found flies very active at night, and even at two in the morning. This is very noticeable along native paths during moonlight, and owing doubtless to some

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peculiar chemotropic property in the odour of the natives, I have seen flies rabid with desire for food under these conditions.

With reference to the use in hospitals, spraying for flies and mosquitos was tried, but the discomfort to the patients, whom it was never possible to move during the operation, was such that the cure was worse than the nuisance.

In conclusion, when the main source of fly infestation, such as butcheries, hide drying sheds, and overflow sections of supplies, was removed to a sufficient distance from the main lines and the absolute cleanliness of all camps insisted on, the fly nuisance soon disappeared.

An important feature of fly control was the prevention of roads, leading near permanent camps, being used for stock and remount purposes, in Dar-es-Salaam, all such animals being stopped at the outskirts, as continual live stock traffic caused a daily reinfestation from far outlying sources, which greatly diminished the value of the work done to free from flies the areas of human habitation.

In this instance there was undoubtedly a good target on each animal, but as time and expediency in moving supplies was the main object, spraying was impracticable, even if means were available, and, as every porter was needed for carrying food and ammunition, out of the question further up the line.

Professor W. J. R. SIMPSON: I agree with Major AUSTEN and the other speakers that the most efficient method of dealing with the fly pest is to attack the flies in their larval stage and remove their breeding places. The sanitarian should pay chief attention to the prevention of the flies rather than to their destruction. When all that can be done in this direction of prevention is accomplished, my experience is that the spray is useful in getting rid of the discomfort and nuisance of the few stray flies that may enter the tent or hut. One's practice in Macedonia was to secure a fairly large area of ground around the hospital, so that passing regiments that were not very particular about sanitation could not encamp in too close proximity to the hospital

Colonel L. G. CUMMINS: I should like to ask, (1) whether any of the larger types of sprays, such as the "Vermorel," have been found useful in fly-destruction; (2) what means Major E. E. AUSTEN has employed to get a fine London fog-like mist of pyrethrum powder diffused through the air of rooms. I agree with Major AUSTEN that the best anti-fly measures were those directed against the breeding of flies.

Major AUSTEN: Colonel CUMMINS's question as to the means by which pyrethrum powder may be distributed through the air of rooms, so as to produce a fog-like mist, can be answered very simply. In default of small bellows of a special type, fitted with a tin container for the powder and purchasable in Rouen in 1915, a piece of muslin six or eight inches square will answer all requirements. All that is necessary is to ladle a few teaspoonfuls of the powder on to the centre of the muslin, screw up the latter with the hand so as to form a bag, and then, having first closed all doors and windows, to walk about the room shaking the bag until the desired effect is produced

Surgeon-Captain P. W. BASSETT-SMITH, R.N.: May I draw attention to the fact that different circumstances alter very much the requirements of treatment. In the open and in the tropics the spray method of destroying flies might give but poor results, yet, speaking from a naval point of view, the method suggested by Professor LEFROY could be used with very great advantage in ships on those stations where flies are a veritable pest at certain seasons, chiefly in the Eastern Mediterranean, Persian Gulf, and Indian and Australian Ports. That the flies are a source of danger as carriers of disease on board is recognised, and many methods have been used more or less successfully to get rid of them, but none appear to have been very satisfactory. Formalin was the best.

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Professor LEFROY: I agree with Major AUSTEN as to the value of fly-sprays in relation to the methods of dealing with flies in war areas. Sanitation is of the first importance. In Mesopotamia, the system of sanitation was one calculated to breed the maximum number of flies in the minimum time; a modification of this was the first necessity. Secondly, poisoning with sweetened sodium-arsenite was effective in Mesopotomia. I am not advocating fly-sprays, but am describing how they had been arrived at, and suggest that their use is limited really to hospitals and private houses, particularly in the tropics, also during the summer in fly-infested villages in England.

With regard to the use of powder as against the extract, the objections to the powder were the want of means of applying it, and the fact that the powder made the room dirty.

I would inform Col. BALFOUR that the castor oil is added only to the pyrethrum-safrol mixture. I have had no personal experience of the castor oil mixture in the tropics, but I have had two letters from Mesopotomia to the effect that the addition of the oil had made the spray effective.

"Osberol" is probably the spray referred to by Col. BALFOUR, and is claimed to be made to the formula of Mr. O'SULLIVAN BEARE. Cinol was believed to be a spirit infusion of pyrethrum made by Keatings.

In answer to Col CUMMINS, I may say that the small sprayer referred to was really an atomising device, and that ordinary sprayers do not atomise the liquids finely enough. A good atomiser for use on a large scale is needed.

The Chairman thanked the reader for his paper, and made a few appropriate remarks.

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AN OUTBREAK OF ITCH DUE TO A PREDACEOUS MITE, OCCURRING IN ENGLAND AMONGST MEN ENGAGED IN UNLOADING COTTON SEED FROM EGYPT.

BY

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The outbreak described in this paper has been referred to by F. C. WILLCOX in his paper published in the *Agricultural Journal of Egypt*, 1915, where he also mentions the occurrence of three cases in Colchester about the same time. But the clinical condition or treatment of the London cases and the experimental work done were not known to the author, and I therefore take this opportunity of placing the facts before this Society.

Towards the end of November, 1913, I was requested to examine four men (stevedores) who refused to complete the unloading of cotton seed, imported from Alexandria in two ships, at the London Docks. Each man complained that, after having worked for about four hours at the cargo, he had become infected with an itch, which extended over his body, caused great irritation, necessitating constant scratching, and prevented him from sleeping at night. Two of the men further asserted that their wives—and one that his children as well—had been attacked by the itch. The men believed that these relatives, who had never been near the cargo, had been infected directly from the workmen themselves. None of the relatives, however, came forward for inspection.

On examination I found that each patient was suffering from an eruption of discrete character. In the worst case the rash extended, including the arms, from the neck to the ankles, while the same condition in patches of varying size, and in lesser degrees of severity, affected the other men. The hands and faces of all were unaffected, and the eruption in each case seemed to begin above the wrists or at the neck, because the papules were most numerous on the forearms and on the chest and upper part of the back.

In the early stages each spot was like a small blister; this soon became pink like a typical papule, which faded on pressure but which returned when the pressure was removed. The spots varied in size from a pin's head to a threepenny-piece. The rash shewed signs of fading where it had first begun, and it seemed more recent (blister-like), where it was undoubtedly spreading, farthest from the areas of early infection. The number and nature of the marks produced by scratching gave evidence of the severe degree of irritation caused.

The fact that the rash was fading in some localities while spreading in others, and that all cases were not equally affected, pointed to a local infection by some skin parasite rather than to a general blood infection; further, the non-inclusion of the head, neck and hands suggested a parasite requiring warmth. There was no constitutional disturbance. Blood examination revealed a slight increase in the number of eosophinile leucocytes; no other abnormality was found and no blood parasites were discovered. The men had suffered from the condition for forty-eight hours when I first saw them. I recommended hot baths, to be followed by sulphur inunction, and a change of clothing till the men's own clothes had been disinfected. After this treatment the irritation ceased, and the rash soon disappeared.

On examining the cargo nothing abnormal was seen on the surface of the cotton

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seed, but through small holes in some of the seeds I noticed a small, pink, living maggot, and, on opening other shrivelled and apparently unhealthy seeds, I found similar maggots in the interior. I collected some of the seed and placed it in a warm room at the London School of Tropical Medicine. On microscopical examination I found creeping over some of the maggots a small mite, which was identified by Colonel ALCOCK as *Pediculoides ventricosus*. Macroscopically the mite could not at first be seen, but, on keeping, it multiplied rapidly, and each infected maggot became covered and obscured by a flour-like coating, somewhat resembling a mould, which consisted of masses of mites, amongst which large pregnant females could be distinguished with the naked eye. Some of the uninfected maggots were collected and kept separately by Colonel ALCOCK, and the adults which emerged were identified by J. HARTLEY DURRANT as *Gelechia gossypiella*.

I placed some of the mites in a watch-glass, which was then adjusted concavity downwards, against the skin of my forearm, and kept in position by adhesive strapping. After about six hours I began to experience marked irritation, and a few spots developed within the circumscribed area, identical in appearance with the spots which I had previously observed on the patients. No mites had been seen on the skin of the patients at my examination. Neither on them nor on myself could I see any signs of burrowing. The irritation and rash are, no doubt, caused by the bite of the insect, and, possibly, also by the numerous bristle-like hairs which protrude from its cuticle.

Since the above observations were made I have served in Egypt, and while at Alexandria I learned that the maggots of *Gelechia gossypiella* are parasites of the cotton plant and its seed in that country, and that an itch, similar to that which I have described, has on rare occasions occurred amongst the native cotton workers, especially the packers. Dr. M. SOROUR has informed me recently that though he knew of the existence of the mite, and the larva of *Gelechia gossypiella* in cotton seed yet he had never seen a case of itch which was attributed to the presence of the former.

The mite, *Pediculoides ventricosus* (NEWPORT), was first discovered at Gravesend, in Kent, where it was found to be parasitic on *Anthophora retusa*. It has since been found in other parts of England, in France, the United States, Mexico, and Egypt. It is known in these countries to be predaceous on many insects and their larvæ, especially on those of the latter which have soft cuticles. In Egypt it is recognised as a parasite of *Galechia gossypiella* (cotton seed moth) and *Galechia cerealella* (grain moth). It is known also to infect some insects found in straw. From an agricultural standpoint it is considered useful in assisting the control of insect pests injurious to grain and seed.

Our earliest information of infection of man by the mite comes from America, where, in an excellent paper by F. M. WEBSTER, the histories of several outbreaks of itch, so caused, are described. Some of these outbreaks occurred on a large scale.

The Tarsonemidae is a family of small mites, the females of which are recognised by the presence of a club-shaped appendage, which projects between the bases of the first and second legs on either side, and also by a pair of conspicuous airholes, or spiracles situated on the ventral surface, just in front of the first pair of legs.

The Tarsonemidae resemble other mites in the arrangement of the legs, the first and second pairs being arranged in juxtaposition to the head, and the third and fourth pairs far away near the hinder end of the body, and also in having long stalked terminal suckers on some of the legs; but the body is softer, and in the female it is not so squat.

Among the Tarsonemidae the genus Pediculoides is recognised by the very distinc head, formed chiefly by the curved and comparatively stout pedipalps.

The species *P. ventricosus* derives its name from the fact that in the pregnant female the lower part of the abdomen becomes swollen to form a large brood-sac. In the latter the embryos develop, and on hatching live within the mother and at her expense, without leaving the brood-sac until fully matured. Finally, they make their exit through the remains of the abdomen of the mother, which they have destroyed during their development.

As the mite is known to infect some of the parasites of straw and different kinds of grain in tropical countries, and as outbreaks of itch have been traced to its agency on several occasions in the United States, and now in this country, it would be well to bear

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its existence in mind as a possible cause of itch occurring amongst people who work in straw or cotton seed, and amongst those who complain of a similar complaint after sleeping on newly-made straw mattresses.

I am indebted to Colonel A. ALCOCK for kind help and criticisms while I was preparing this paper.

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1. WEBSTER, F. M., United States Department of Agriculture (Bureau of Entomology), 1910, April 23rd.
2. WILLCOX, F. C., "The Predaceous mite *Pediculoides ventricosus* (NEWPORT)." *Agric. Jl. of Egypt.* Cairo. No. I. June, 1914. Pp. 17-51.

DEMONSTRATION.

The following microscopical specimens, stained by the iron hæmatoxylin method after fixation in Schaudinn's fluid, were shewn by Dr. F. W. O'CONNOR :—

1. *Entamœba histolytica* cyst, containing 8 nuclei. The cyst measures 16·5 μ. The nuclei are typically those seen in *E. histolytica* cysts, with small central karyosomes and chromatin heaped in crescentic formation on the nuclear ring. The stool of the patient was examined on 44 occasions, and during this period no *E. coli* cysts were seen.

2. *E. coli*, free form, containing 2 lamblia cysts included in vacuoles.

3. *E. coli*, free form, with an *E. histolytica* cyst included. The cyst contains 4 nuclei and chromatoid bodies.

4. *E. coli*, free form, with an oöcyst of *Isospora hominis* included. The oöcyst measures 28 × 14 μ, and the ends bulge the amœba at opposite poles. In the fresh specimen 10 different *E. coli* were seen to possess a similar inclusion.

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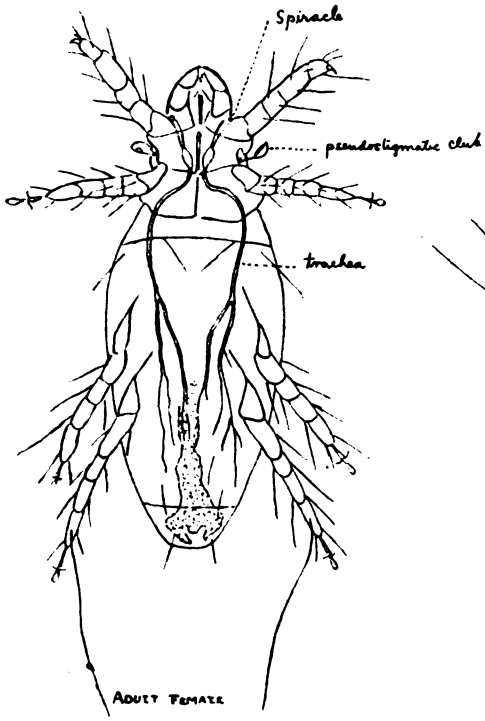
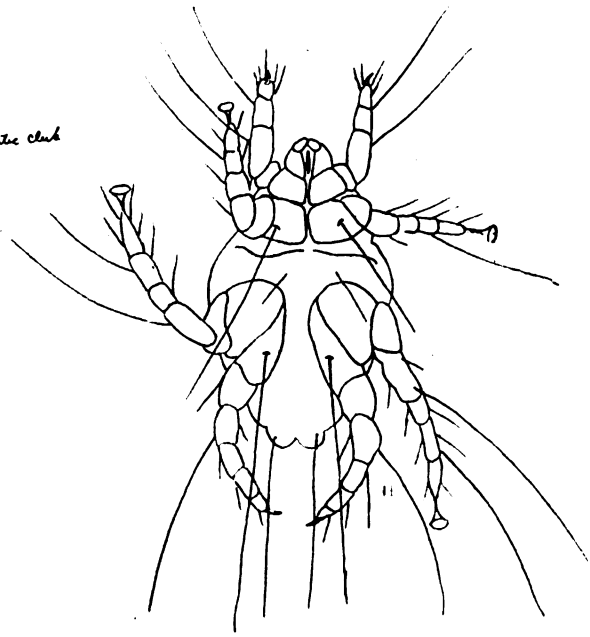


ADULT FEMALE



MALE.

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CORRESPONDENCE.

Prestea, via Seccondee, West Africa, Gold Coast.

April 9th, 1919.

DEAR DR. LOW,

In the TRANSACTIONS of October 18th, 1918, under "Notes and Comments," I see that Major HUGH S. STANNUS has recorded some corrections. In the last paragraph but one he states, "I should like to add that rickets is unknown in all Central African races I have seen."

Until 1916 I should have agreed with him, but in that year I had a case which was of interest because it appeared to me to be rickets. The notes I then wrote about the case were as follows :—

> Belgian Congo, Kasai Basin, 1916.—Boy, aged about 12 years, deformed from his earliest memory. Typical rachitic rosary, pigeon breast, Harrison's groove. Lower limbs bowed, periosteal thickening, epiphyseal enlargements. Upper limbs, bones of forearm bent, irregular in outline, edges rounded, marked epiphyseal thickenings at ends of ulna and radius. General debility. Not as well built as boys of his age and height. No evidence of congenital syphilis.

Yours very sincerely,

W. E. MASTERS,

M.D. (Brux.), M.R.C.S.

The Secretaries, Society of Tropical Medicine,

11, Chandos Street, Cavendish Square, W. 1.

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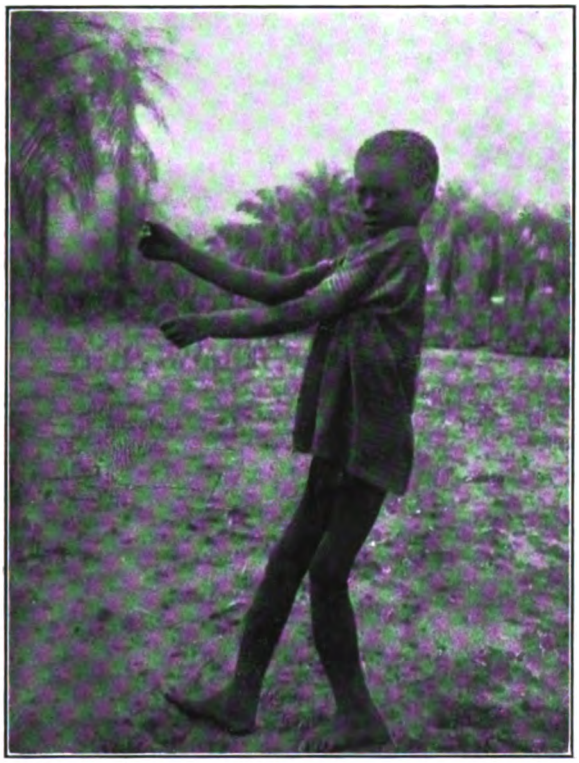

Case of Rickets in African Boy.

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TRANSACTIONS

OF THE

SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

JUNE 20TH, 1919.

VOLUME XIII. No. 2.

Proceedings of a Meeting of the Society held on Friday, June 20th, 1919, at 5.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Surgeon-General Sir DAVID BRUCE, K.C.B., F.R.S. (*President*), in the Chair.

ANNUAL MEETING.

The Chairman stated that the Annual Report and Statement of Accounts, as passed by the Auditors, were before the Meeting. These were duly proposed and seconded, and adopted without discussion.

The result of the ballot for President, Vice-President and Committee for the ensuing two years was announced as follows :—

President :

Professor W. J. R. SIMPSON, C.M.G.

Vice-President :

Sir JAMES CANTLIE, K B.E.

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Council:

Major E. E. AUSTEN, D.S.O., F.Z.S., R.A.M.C.
Dr. A. G. BAGSHAWE, C.M.G.
Dr. ANDREW BALFOUR, C.B., C.M.G.
Surgeon-Capt. P. W. BASSETT-SMITH, C.B., C.M.G., R.N.
Sir JOHN ROSE BRADFORD, K.C.M.G., C.B., F.R.S.
Surgeon-Capt. G. T. COLLINGWOOD, M.V.O., R.N.
Colonel S. LYLE CUMMINS, C.M.G., R.A.M.C.
Professor CLIFFORD DOBELL, M.A., F.R.S.
Dr. C. F. HARFORD.
Dr. W. HARTIGAN.
Lieut.-Col. S. P. JAMES, I.M.S. (retd.).
Colonel W. G. KING, C.I.E., I.M.S. (retd.).
Professor R. T. LEIPER, M.D., D.Sc.
Dr. G. C. LOW.
Sir STEWART STOCKMAN, M.R.C.V.S.
Dr. C. J. MARTIN, C.M.G., F.R.S.
Dr. H. B. NEWHAM, C.M.G.
Dr. W. T. PROUT, C.M.G.
Professor J. W. W. STEPHENS, M.D.
Dr. C. M. WENYON, C.M.G.

Sir DAVID BRUCE: It is now my duty to vacate this chair and give place to our new President, Professor SIMPSON. I have sat in it since June, 1916, so that I have had a double portion. The last years have been difficult ones for the Society on account of the occurrence of the European War, in which England was forced to take a hand, and which we all sincerely trust has now come to an end. On this account we have not been able to keep up our membership—633 in 1914 and now 566.

In regard to the advance in knowledge of tropical diseases during these four years, naturally, under the circumstances, I have not a very flattering tale to tell. In 1916, perhaps the most interesting discussion was on "Combined Vaccinations," introduced by Professor CASTELLANI. At that time there was some doubt as to the wisdom of adding paratyphoid A and B to the existing typhoid vaccine; but this was shortly afterwards done in the Army, and, as I understand, with satisfactory results. It will, however, be a long time before we are in possession of the complete figures. I may say, in passing, that Army figures are often hard to interpret. There may be a hesitation sometimes in diagnosing such diseases as typhoid and dysentery on account of the implied reflection. In this way such terms as P.U.O. and diarrhœa may have unduly forced themselves on the attention. This method of preventing disease by vaccines may be permissible in war, but I hope that in times of peace some more radical method will be striven for.

In the same year, Dr. Low introduced the subject of the use of antimony in the treatment of certain tropical diseases. It would appear that this drug is extending its usefulness.

In 1916, Dr. HAROLD SCOTT also, in a dramatic way, advanced our knowledge of the vomiting sickness of Jamaica.

In 1917, Dr. HARRIETTE CHICK and Miss HUME described some of the work they were carrying out at the Lister Institute on the vitamines concerned in the prevention of scurvy and beriberi. This work, begun as war-work, has been continued, and must prove of immense use in public health.

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In 1918, an important discussion on the treatment of malaria was introduced by Sir RONALD ROSS, who was followed by JAMES, MACDONALD, STEPHENS and others. This is an important subject and ought to be persistently pursued until definite conclusions can be laid down as to quinine as a prophylactic and a therapeutic agent.

In May, 1918, another interesting subject was brought up by Major BYAM and his colleagues at the Hampstead Military Hospital. This is an example of team work, BYAM having associated with him some eight workers who take up different sections of the work. Trench fever is said to belong to the group of filter passing viruses, which are attracting so much attention at the present time. It has been abundantly proved that the disease is carried from man to man by the louse, but up to the present the team have not succeeded in cultivating the virus and giving rise by the sub-cultures to the disease in a healthy man, as has been claimed by BRADFORD, BASHFORD and WILSON in France. It is hoped to test this observation within the next few weeks. If confirmation is obtained it would appear that we are at the beginning of a decided advance in our knowledge of several important diseases.

I have now the honour to vacate the chair in favour of the newly elected President, Professor SIMPSON.

Professor SIMPSON then took the Chair.

The PRESIDENT, after expressing his appreciation of the honour done him, called for the proposal of Auditors.

The names of Dr. BEDDOES and Dr. BAHR were proposed, and these gentlemen were duly elected.

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COMPLEMENT DEVIATION IN MALARIA AND THE QUESTION OF THE INFLUENCE OF MALARIA ON THE WASSERMAN REACTION.

BY

J. GORDON THOMSON, M.A., M.B., CH.B.

Protozoologist to the London School of Tropical Medicine.

The object of these researches was to ascertain two points, namely, the diagnosis of malaria by the deviation of complement when using a specific antigen, and the influence of malaria on the Wasserman test as performed in the diagnosis of syphilis. The value of some other pathological method to aid in the diagnosis of malaria, and to help in estimating the effect of treatment, is recognised by all to be of great importance. Patients from the tropics often state that they have had malaria, but when parasites cannot be found in the peripheral blood, it is difficult to be absolutely certain that their fever was really due to this.

The great value of the Wasserman test in the diagnosis of syphilis is well known to you all, and the fact that so many of our troops have been infected with malaria during their service abroad has led to great confusion, especially when it is stated in practically all the text-books that malaria must be excluded when a blood serum gives a positive result. In Egypt, where I performed the Wasserman test on the serums of many thousands of cases to aid in the diagnosis of syphilis, the question of malaria as a complicating factor was a serious one, for if the patient gave a malarial history and a positive Wasserman, we were, when the clinical signs of syphilis were absent, always in doubt whether the patient had syphilis or not. This resulted in some cases with a positive Wasserman being left untreated.

In a series of experiments done by Mr. CLAUDE H. MILLS, Specialist attached to Rochester Row Military Hospital, and myself (1919), I am glad to say we have been able to settle this disputed point, to which I shall refer later in this paper.

COMPLEMENT DEVIATION AS A TEST FOR CASES OF MALARIA.

In order to successfully obtain fixation of the complement in malaria cases it is necessary to obtain a specific antigen. In doing this I have prepared one from cultures of malarial parasites (1913). The chief purpose of this research was to enable us to diagnose recoveries from malaria where no parasites could be found in the peripheral blood, and also to estimate the effect of treatment by quinine.

As the fixation of complement depends on the presence of antigen and its specific antibody, the success of the test depends on the actual presence of antibody in a malarial patient; if that is so, and we can obtain the specific antigen, the test should be possible.

The difficulty in the past arose in not being able to produce a satisfactory specific antigen. Alcoholic extracts of the spleen of malarial cases have proved unsatisfactory. We want for an antigen an extract of the bodies of malarial parasites; the nearest approach so far towards obtaining this is by using the following technique. A culture of

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malarial parasites is obtained; the supernatant fluid is pipetted off, and an excess of distilled water is added to the remaining corpuscles and parasites. This is shaken up, and so the corpuscles are laked. Place the tube in a centrifuge and drive down the parasites and broken up envelopes of the red cells. Repeat this process as often as is necessary to remove all the hæmoglobin. Finally, pipette off all the supernatant water, and if the sediment is examined it will be found to consist of practically a solid mass of malarial parasites, mixed with leucocytes, and empty envelopes of red-blood cells. Dissolve this sediment in N_{10} NaOH, and then neutralise with dilute HCl. This gives us a neutral solution of malarial parasites with some débris of corpuscles and leucocytes. Dilute with normal saline until by tests we find it has no anticomplementary action *per se*, and use this as antigen. It seems unnecessary to give the full technique used, as this has already been given in the *British Medical Journal* (1919), *The Proceedings of the Royal Society of Medicine* (1919), and full details are being published by Sir RONALD ROSS in a War Office publication now in the press.

I should like simply to state that by using an antigen so prepared, it is possible to obtain fixation of the complement in acute and chronic cases of malaria. The test is rather a delicate one, but $2\frac{1}{2}$, 3, and $3\frac{1}{2}$ minimum hæmolytic doses have been absorbed, and this proves the presence of antibody in cases of malaria.

The test must be done by a careful and experienced observer, or the results will be worthless and confusing.

I shall now give a synopsis of experiments actually done:—

(*a*) Preliminary experiments were performed with the serums of known benign tertian cases, all with *Plasmodium vivax* present in the peripheral bloodstream, using an antigen prepared from a single strain of *P. vivax*. In this series eleven cases gave a strongly positive result, seven gave only weakly positive results, and one was entirely negative. This latter case had a rigor the day before, and whether the serum was deficient in antibody, or the antigen used non-specific to this particular parasite, we were unable to determine.

(*b*) Experiments were conducted in order to determine if separate strains of parasites reacted differently to various antigens. This was a difficult point to determine, as the real cause of the different results obtained might possibly be due to slight differences in the preparation of the antigen used. Some antigens prepared were quite useless, owing to their being too anticomplementary, or producing only negative results.

(*c*) Experiments were made for the purpose of determining if specific antigens could be obtained for benign and malignant tertian malaria.

Here the difficulty of mixed infections arises, as we can never be quite certain that a patient has not a double infection. Certainly, some cases only react to their specific antigen, but there may be a group reaction as well.

(*d*) When using antigen prepared from the spleen of a malignant tertian malaria, and an antigen from a culture of *P. vivax*, certain cases gave a specific reaction to the infection found, but in other cases a positive reaction was obtained when using either antigen.

(*e*) A compound antigen made from ten different strains of parasites, all *P. vivax*, was used to test a number of cases of benign and malignant tertian malaria. Five cases of malignant tertian malaria gave a positive result, indicating either a group reaction or an undiagnosed double infection, which is probably commoner than we suspect.

(*f*) A large number of cases of malaria were examined, using a compound antigen, and these were tested at every stage of the disease, and after all methods of treatment had been administered. It was found that individual cases of malaria varied greatly in their reaction to quinine. Some reacted quickly to treatment and others resisted the effect of the drug.

The general conclusions shewed that treatment needed to be prolonged in most cases, in order to render the serum negative. No doubt it will be shewn that the initial attack, treated promptly and thoroughly, will give the best results.

(*g*) Finally, a series of experiments was conducted co-relating the large mononuclear counts with the complement deviation, and in practically all cases it was shewn

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that the mononuclear count was a useful factor in diagnosis. The charts shewn by the lantern will illustrate these points.

The Influence of Malaria on the Wasserman Reaction used in the Diagnosis of Syphilis.

We now come to the disputed question, "Does malaria give a positive Wasserman reaction?" This is now a most important point in this country when so many soldiers have been in malarial countries, and especially now we are having formed all over the country venereal cliniques. It is also of importance medico-legally. In conjunction with Mr. Claude H. Mills, a series of 130 malarial cases were tested by the routine standard Wasserman, as used at Rochester Row for the diagnosis of syphilis.

The sera of these malarial cases were examined at every stage of the disease, and only eight gave a positive result. A careful clinical examination by Mr. Mills demonstrated in each positive case the presence of syphilis (1919). We carefully went into the literature on this subject, and were forced to conclude that care had not been taken to exclude syphilis, the coexistence of which in any given series of cases is by no means rare. Unreliable technique seems also to have played a part in causing confusion.

A positive Wasserman reaction, therefore, in a patient either suffering from malaria or with a history of malaria, indicates syphilis, provided the technique is correct, and a standard Wasserman method does not give a positive reaction in malaria at any stage of the disease.

Conclusions.

In summing up the results of these investigations, we are able to state that it is possible to obtain a deviation of the complement in cases of malaria by using a specific antigen, and this may prove of value in the diagnosis of doubtful cases. As the reaction is a weak one, and therefore liable to error, it must be carefully controlled against known negative serums, and the results weighed carefully with all the other clinical evidences.

We hope that researches along these lines will lead to better technique and a more reliable antigen.

The fact that malaria does not give a positive Wasserman reaction is of great importance, as until the present research the clinician and the pathologist were always in doubt about this.

Finally, I wish to tender my grateful thanks to Sir Ronald Ross, K.C.B., Brevet-Colonel Harrison, Mr. Claude H. Mills, and Dr. David Thomson for all their kindness and help during these researches.

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Discussion.

Col. Harvey: I should like to ask if Dr. Thomson has found his test of value in the differential diagnosis of the more obscure cases of fever, in which, perhaps, the presence of malaria had afterwards been demonstrated by relapse and the appearance of parasites in the peripheral blood.

Dr. Philip Bahr: I wish to congratulate Dr. Gordon Thomson most heartily on this useful and painstaking piece of research. As one of his late colleagues in Egypt, I have been watching with interest the results of his labours. I am especially interested in the difficulty in malaria of obtaining a satisfactory antigen. Perhaps some lessons may be learned from the researches on the complement-fixation in bilharziasis of my friend Lt.-Col. Fairley, A.A.M.C., with whom I have had the pleasure of being associated from time to time during the last two-and-a-half years. I am certain that there is a great future before complement-fixation diagnosis in helminthic as well as in protozoal disease, for, after all, the spirochæte of relapsing fever and the malaria parasite are such "elusive pimpernels."

In bilharziasis a most satisfactory antigen, which will absorb six M.H.D.'s of complement, is obtained from the liver of the snail, *Planorbis boissyi*, infected with the cercariæ of *Bilharzia mansoni*. It is probably a proteid body, and can be extracted by macerating the cercariæ in normal saline. It is a more specific test than is the Wasserman in syphilis, in that the antigen is absolutely specific for this infection and is not a lecithin body as it is in the classical Wasserman test. It is, however, a group reaction, in so far that antigen obtained from *mansoni* cercariæ will react with *hæmatobia* sera, and possibly with *japonica* as well. The antigen obtained from the larval cercariæ is much more satisfactory than that obtained from the adult parasites. Alcoholic extracts of the infected snail's liver are not so specific, in that they give a positive reaction with syphilitic sera. The difficulty at present is keeping the antigen indefinitely, for saline extracts even if preserved with phenol develop an anti-complementary tendency on keeping.

Now all this has, I think, a bearing on the complement-fixation in malaria. Having obtained a specific substance from the invertebrate host in one disease, it might be possible to do so in others. I realize there are great difficulties in the way, but it might be possible to obtain as strong and specific a substance from the oöcysts of the malaria parasite in the anopheles for malaria, and of filarial larvæ in the thorax of appropriate mosquitoes for filariasis, as Col. Fairley has proved it is possible to obtain from the liver of infected *Planorbis* for bilharziasis. I would merely like to throw this out as a suggestion for further work.

There is one point of interest which may have a bearing on Dr. Thomson's work, namely that the sera of severe cases of malaria failed to give a positive result. Now monkeys overwhelmed with bilharzia infection in which there is no cellular-humoral response (as for instance, absence of eosinophilia) give a negative reaction, but those which are resisting the injection well give a positive. It may be then that the sera of mild cases of malaria will give a stronger reaction than those overwhelmed with the toxins of the parasite.

I wish we had been able to preserve malaria spleens for the manufacture of antigen for Dr. Thomson. There was a time, I am sorry to say, when we were overwhelmed with them in Palestine. I am sure they were rich in antigen, for they were absolutely swarming with subtertian parasites. The difficulties of obtaining any such in this country must now be considerable.

Once more I would like to congratulate the author on his paper, and the succinct and clear manner he has put it before the Society.

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Sir DAVID BRUCE: I have recently met an American, Dr. WATSON, who said that he had carried out a deviation test with dourine in his country, and had found it quite practical.

Dr. J. G. THOMSON: With reference to Col. HARVEY'S remarks, I beg to state, that the test, when positive, in doubtful cases ought to be carefully weighed with all the clinical evidence available, or errors are liable to occur in diagnosis. I have to thank Dr. BAHR for his kind remarks, and corroborate his statements that researches in complement deviation in protozoal and helminthic infections are likely to be of great value in the future. I am interested to hear what Sir DAVID BRUCE says about dourine, and I intend to conduct a series of researches in order to find if the complement deviation test can be used in the diagnosis of trypanosomiasis.

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ON THE OCCURRENCE OF *CŒNURUS GLOMERATUS* IN MAN IN WEST AFRICA.

By M. TURNER, B.Sc., and R. T. LEIPER, M.D., D.Sc.,
Helminthological Department, London School of Tropical Medicine.

In May of this year, Dr. W. B. Johnson, of the West African Medical Staff, supplemented previous valuable gifts to the collections of the London School of Tropical Medicine by presenting to the Helminthological Department a cystic tapeworm, found by him in a tumour in a native of Kaduna, Northern Nigeria.

The tumour had been excised from an intercostal muscle. Dr. Johnson had duly recognised its parasitic nature, and, after incising the cyst, had cleared and mounted the lining membrane to shew the scolices.

Cystic tapeworms have been recorded on several occasions from the subcutaneous tissues of man. Almost without exception they have proved themselves to be examples of *Cysticercus cellulosæ*. Early this year Fontan recorded a first case of infection with *Cysticercus bovis*. In these instances the cysts have resulted, most probably, from auto-infection, man being the normal host of the adult parasites, *Tænia solium* or *Tænia saginata*.

Dr. Johnson's cyst differs from the preceding, for it contains numerous "buds" or invaginated "heads," *i.e.*, is a Cœnurus larva. There are only three references in literature to the occurrence of the Cœnurus in man. Mention is made of this form in *Dissertationes anatomicæ metodo synthetica*, published by Rolfinke in 1656. Klencke, in 1844, stated that he had seen a case. No details are given, and these records have always been regarded as of doubtful validity. In 1913 Brumpt described and figured the first well-authenticated case in the third edition of his *Précis de Parasitologie*. The cyst occupied one of the lateral ventricles of the brain, and presented, according to Brumpt, the same characters as the *Cœnurus cerebralis* of sheep.

Our specimen is therefore the first Cœnurus found in the subcutaneous tissues of man.

Six different species of Cœnurus larvæ have been differentiated up to the present time, viz., *C. cerebralis* in sheep, *C. gaigeri* in goats, *C. serialis* in rabbits, *C. lemuris* in a lemur, *C. polytuberculosus* in a jerboa, and *C glomeratus* in a gerbille.

The adults of the first three alone are known to occur in the dog. The other Cœnurus larvæ are rare, a single infection with each being recorded.

The various species of Cœnurus can be best distinguished by the size and shape of the hooks. By its hooks *C. glomeratus* can be easily differentiated. The handle of the hook is shortened and shows a characteristic notch. The blade is strongly curved. In these respects Dr. Johnson's specimen conforms exactly with the type specimen of *C. glomeratus* obtained by Railliet and Henry in 1915, from the gerbille in Tunis.

Description of the parasite.—The tumour from which the cyst was removed measures 2 cms. by 1 cm. The wall of the cyst is very delicate and transparent, containing few calcareous bodies.

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Projecting into the cavity of the cyst are about thirty-five buds or invaginated heads, which are so crowded with calcareous bodies as to be almost opaque.

The buds measure from 110μ to 120μ by 35μ to 45μ. Each bud contains an inverted scolex, bearing four suckers and a double crown of hooks. The suckers have a diameter of 130μ, and as a rule the hooks number thirty-two, viz., sixteen large and sixteen small hooks.

There is a certain amount of variation in the size and shape of the hooks in the group Cœnurus as in the genus Tænia. Usually, however, the hooks have a prolonged and sinuous handle from which the guard projects abruptly. In *C. glomeratus* the handle is short, stout and conical, merging gradually into the guard. From the broad base so formed a strongly developed and well-curved blade springs. RAILLIET and HENRY found that, in their specimens from the gerbille, the number of hooks varied from eighteen to thirty-four, that the large hooks measured from 96μ to 105μ in length, and that the small hooks measured from 58μ to 65μ. The highest figures given by them applied to the best developed hooks. These measurements are less than those obtained by us from Dr. JOHNSON's specimen, in which the large hooks measure from 90μ to 110μ, and the small hooks from 65μ to 70μ.

These differences are within the range of variation found by us to occur in the hooks of various buds taken from a single cyst of *C. cerebralis*.

In RAILLIET and HENRY's specimen the guard of the small hook is slightly bilobed, and the handle of the large hooks is characteristically notched on the dorsal or convex border, at a point one-seventh of the total length of the hook from the end of the handle. In these significant features our specimen tallies exactly.

The occurrence of *C. glomeratus* in man is apparently the result of accidental contamination with fæces from a carnivore harbouring the adult tapeworm, which, so far, has not been described.

Throughout this paper we have used the old descriptive term Cœnurus for the polycephalous larvæ, found in the genus Tænia, in preference to *Multiceps*, which has recently been revived as a generic name We are not yet persuaded, on the facts available, that the occurrence of several scolices in a larval cyst is, of itself, a character of generic significance.

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ENTERIC FEVER AND PROPHYLACTIC INOCULATION IN BRITISH GUIANA.

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It would be difficult to exaggerate the importance of enteric fever as a cause of ill-health and death among the inhabitants of British Guiana. In Georgetown, the capital town, it is endemic with periodic effervescences, and among the 50,000 odd inhabitants of the town there were, in 1914, 154; in 1915, 196; in 1916, 190; in 1917, 272; and in 1918, up to October 31st, 206 notifications of enteric fever.[2]

Exhaustive investigations have, therefore, been made into the causes and the available means of prevention of the disease in this colony.

Koch first pointed out, as Harvey[1] reminds us, that the ultimate source of all infection is the infected person, whom he divides into three classes:—

1. Persons actually suffering from enteric, who are excreting the bacillus in their fæces, or urine, or both: *a*, Recognised; *b*, Unrecognised.

2. Persons who have recently passed through an attack of enteric, and are excreting the bacillus in their fæces, or urine or in both: Temporary carriers.

3. Persons who have had an attack of enteric fever more than six months previously, and are still excreting the bacillus in their fæces, or urine, or in both: Chronic carriers.

Harvey, in his articles in the *Journal of the Royal Army Medical Corps* on the Causation and Prevention of Enteric in Military Service, concludes that, in military service, Class 2, the temporary carriers, are the most prolific cause, followed by Class 1 *b*, and then by Class 3. He adds, however, that the carrier in civil life is not the danger to the community that infected persons are in the army.

Now, the conditions under which the civil community live are subject to the widest variations. In Georgetown the vast majority of the cases of enteric are drawn from the poorer classes, the blacks and East Indians, who live under conditions very nearly akin to barrack-life in its most insanitary form, as we shall presently see, and, if Harvey's conclusions are correct, it would be the case that the carrier in Georgetown is every whit as dangerous as the army carrier.

What are the conditions of life among the poorer classes in this city? The great majority of its inhabitants belong to the black and East Indian races, and are persons belonging to the lower classes, and, therefore, in common with these classes in every other part of the world, of a very low standard of cleanliness. Most of them live in dwellings known as ranges, that is to say, wooden houses of one flat, consisting of a long row of single rooms, each of which is often divided by a screen, locally known as a "part-off," into two parts, one serving as a living and the other as a sleeping-room. Each range may consist of a number of rooms varying from two rooms to nine or ten, and each room almost invariably houses more than one person, often a family of six or more.

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A yard may contain several of these ranges, and perhaps some small two-roomed cottages, housing over thirty inhabitants, and provided with two closets, each with one or two compartments, draining into a single cess-pit, and ministering to the wants of the whole of the thirty odd inhabitants.

The closet is frequently to be found stained with fæces and moistened with urine; toilet-paper consists of scraps of old newspapers, rags, etc., always inadequate, and, especially in the case of children, may be dispensed with altogether Washing of the hands after the act of defæcation is regarded as a luxury and is but seldom indulged in.

Moreover, these dwellings are not confined to one or two quarters, but are indiscriminately distributed throughout the town; some excellent specimens are to be found in the near neighbourhood of Government House.

To add to the dangers inherent in these conditions, these classes are notoriously backward, by reason either of pecuniary difficulties or of pressure of time, in seeking medical advice. Thus, in an analysis made by the writer of fifty-five cases admitted to the Public Hospital, Georgetown, in 1915 and 1916, in which the duration of the disease was plainly established either in post-mortem lesions or such signs as intestinal hæmorrhage, it was found that no fewer that eighteen, or 33 per cent., did not even take to their beds until early in the second week of the attack. It has to be remembered, too, that the rose-spots are not available for diagnosis among the dark-skinned races, so that, even if medical advice is sought, it may be some days before the diagnosis is established, especially as the Widal reaction is not available before the end of the first week of the disease.

When one realises that there were notified in Georgetown in one year over 150 cases, so large a proportion of which were, during at least one week of the infective stage of their illness, going about their ordinary duties undiagnosed, there will be no difficulty in understanding how potent a factor this class probably is in the spread of enteric.

To obtain some evidence that this class is largely concerned, searching enquiries were made into the histories of patients admitted into the isolation ward for enteric cases in the Public Hospital, Georgetown, from August, 1916, onwards.

A few typical cases are appended :—

W.W. contracted the disease about the middle of August, 1916, and left hospital on the 23rd of September; I.W., a relative living in the same house, contracted the disease about the beginning of September. Probably carrier Class 1.

B.M. was discharged from the typhoid ward on October 3rd, 1916; W.M., father, contracted the disease about the beginning of the same month, that is to say, incubation period appears to have started about that date. ? Class 2.

W.M. was discharged from the isolation ward on October 6th, 1916; B.M., daughter of W.M., contracted the disease in February, 1917, ? Class 2; A.M., brother of the former, contracted the disease about three weeks later. ? Class 1 or Class 2.

E.H. contracted typhoid about the middle of October, 1916; H.H., child of E.H., contracted typhoid about the middle of January, 1917. ? Class No. 2; F.H., brother of the latter, contracted the disease two weeks later, simultaneously with M.H., sister, while N.H., another sister, developed the disease three weeks later still. Probably all Class 1 or Class 2. H.C. and K.C., mother and daughter, developed typhoid in December, 1916; A.C., another child of H.C., contracted typhoid about two weeks after the discharge of the first two. Probably Class 2. Similar cases are the family of K., five members, all with a paratyphoid A. infection, the family of P. and of J., with a strong probability of infection by Class 1 or Class 2 of carrier, all telling the same story with monotonous repetition.

These cases of familial infection, in fact, abound.

Class 2, the temporary carrier is, again, a fertile source of infection.

From the stools of thirty-three enteric convalescents just before their discharge from the isolation ward, *Bacillus typhosus* was isolated ten times, and *B. paratyphosus* once, using the method adopted by STOKES and CLARKE.[3]

As to Class 3, the chronic carriers, it goes without saying that, in a town such as this, where enteric is always with us, the chronic carrier must abound.

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The writer investigated two small localised epidemics, in which all the evidence pointed to one of this class, though the bacillus was never recovered from the excreta of the suspect before his disappearance; the serum, in both cases, agglutinated *B. typhosus* in a dilution of 1 in 100 in less than half an-hour.

One confessed to having had enteric four years previously; the other strenuously denied ever having had it; both objected to examination of the excreta and incontinently vanished after one examination. These investigations shewed that Class 2 and Class 1 *b*, and to a less extent, Class 3, probably acted as reservoirs for the enteric organisms in the town.

Enquiry was then directed towards ascertaining the ordinary channels of infection through which the bacillus passed to the prospective patient under local conditions.

This disease, as we have said, is endemic, but at certain periods each year the number of cases shews a decided and alarming increase.

From year to year, since 1912, when notification was first enforced, there has been but slight variation. The largest number of cases was in 1913, when there were 254; this was also the driest year of the series, but in 1914 the number of cases was the smallest of the series, although there were 12 inches less of rain than in 1916 and 8 less than in 1915.

It cannot, therefore, be said that the number of cases in a year has any relation to the dryness of that year.

Further, with regard to the temperature, ROWLAND,[4] who investigated the relationship between rainfall, average maximum and average minimum temperatures on the one hand, and the number of cases of enteric admitted to the Public Hospital, Georgetown, was unable to trace any relationship between them.

With regard to drinking-water supply in Georgetown, it is almost entirely derived from rain-water, which is stored in wooden vats and cisterns; if, therefore, drinking-water played any large part in the spread of enteric, this would be reflected in the corresponding curves. Further, one would expect to find epidemics shewing the special features displayed by the disease when derived from such a source; for example, practically simultaneous occurrence of a group of cases in a yard where all the inhabitants drew their supply from the same vat. Such features are absent from the local epidemics.

Moreover, at a time when enteric was at its height in the town, repeated bacteriological examinations of vat-water from yards containing infected persons failed to reveal any suspicion of exceptional fæcal contamination, nor were enteric organisms ever isolated, though this was hardly a matter for surprise.

The milk-supply has been found to be implicated in one or two isolated cases by WISE,[5] but here, again, one can point to few cases in which such an origin seems at all probable.

In fact, it may be said with certainty that there is no particle of evidence that the prevalence of the disease in this country is associated in any appreciable degree with such agencies as the drinking-water supply, or such foods as milk, shrimps, or shell-fish of any description.

Flies must play but a small part, for, whatever may be the difficulties with which sanitarians have to cope in Georgetown, it cannot be said with any truth that the house-fly is one of any magnitude; in fact, considering the sanitary conditions, we are a remarkably fly-free town; long may we remain so!

With regard to the contamination of the soil, however, there is some evidence, it has been considered, of a connection between this and the spread of the disease.

MINETT,[6] in a review of the causes of enteric, observes that all large towns must abolish the privy and open cess-pit system at whatever cost; otherwise to expect a reduction in the typhoid rate is hopeless. Others have said or implied that the installation of a water-carriage system of sewage disposal will at once bring about a reduction in the enteric rate.

Now, these statements must mean either that such a change will lessen the influence of the carrier as carrier, or else that, in the opinion of the writers, there is some inherent

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defect in the cess-pit system which has the effect of rendering it a powerful intermediary in spreading infection.

Since the former can only be the consequence of a change in his habits, the latter is the only alternative we need consider, and this supposed defect is obviously bound up with the question of the vitality of the typhoid and paratyphoid organisms in the soil, in sewage and in air.

Taking, first of all, the soil, HARVEY,[1] to determine the vitality of the bacillus when voided directly on the ground, made a carrier (urinary) micturate on the earth, and found that six hours after pollution the bacillus was easily recovered from the washings of ½-gram of soil, but thirty hours later could not be found. He concludes from this and other experiments that the surface soil is dangerous for some hours, but is no longer so when dry enough to be blown about as dust.

As to sewage, it is generally recognised that the survival of the bacillus in sewage is dependent on the multiplication, or otherwise, of *B. coli* and other organisms. There is, however, another organism in this colony which, it would appear, deserves to be taken into account in this connection ; I refer to *B. pyocyaneus.*

The writer had been struck by the frequency with which this organism is to be isolated from water-supplies, fæces, pathological conditions such as abscesses, osteomyelitis, etc., in Georgetown. MINETT and DUNCAN[7] have published a paper dealing with its pathogenicity in this colony. A series of experiments was performed by him in 1915 with regard to antagonism between this organism and the typhoid and paratyphoid bacilli on the same lines as some performed by COLEBROOKE in the case of the meningococcus.[8] The results may be briefly set down here. There was found to be no antagonism between *B. typhosus* or *paratyphosus* A or B and *pyocyaneus*, such as COLEBROOKE finds between pneumococcus and meningococcus, nor was inhibitory action anything but slight between *B. coli* and the organisms of the enteric group. The truth appeared to be that both *B. pyocyaneus* and *B. coli* at room temperature grow far more rapidly and profusely than these organisms, and rapidly overgrow them, generally in two or three days.

As to the vitality of these organisms in water, one may quote the findings of HOUSTON[9] given in the Harben lectures of 1916, that "uncultivated typhoid bacilli died in Thames water generally in the first week, but rarely as late as the second and third."

It will be seen, therefore, that it is impossible to eliminate soil-contamination as a factor in the spread of infection, but we may say that the danger in Georgetown is mitigated by the fact that the atmospheric conditions are practically always favourable to the multiplication of *B. coli*, *B. pyocyaneus*, and other organisms in sewage antagonistic to the enteric group of organisms, unless we assume the possibility of transmutation of *B. coli* under certain conditions into organisms of that group.

In a special report to the Surgeon-General the results of these enquiries were embodied and among the measures, it was recommended that the use of prophylactic inoculation should be largely extended.

In July, 1917, accordingly, inoculations began to be made free of cost to those who wished, the vaccine being prepared in this laboratory, first from strains kindly sent to me by Professor LEITH, of Birmingham, and later from the well-known Rawlings strain, used so largely in the U.S.A., *B. typhosus* and *B. paratyphosus* A and B being included in the vaccine.

Two inoculations are given to each person, the interval being ten days, and the adult dosage 1,000 millions of bacilli at the first inoculation, and 2,000 millions at the second, each dose being made up of *B. typhosus* and *B. paratyphosus* A and B one part each.

The time has arrived when it is possible to take stock of the results of this—as far as I am aware the first experiment in anti-typhoid inoculation on so large a scale in a civilian community.

Other measures included, as regards the city of Georgetown, the strict isolation of enteric cases and the testing of fæces and urine on three separate occasions by this department for organisms of the enteric group before discharge.

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The method used has been that recommended by BROWNING and his fellow-workers.[10]

There are in the colony three well-marked centres of infection:—The city of Georgetown in particular; a group of sugar estates on the East Bank of the Demerara River; and a group of sugar estates and villages on the coast-line. These three centres were responsible jointly for 299 of the 364 cases notified from all parts of the colony in 1916, Georgetown heading the list with 190, the East Coast coming next with 58, and the East Bank furnishing 51 cases.

From the latter months of 1915, inoculation was energetically undertaken by the Government Medical Officer of the district on several of the East Coast estates; latterly this has been extended to most of these estates, and inoculation has also been extensively resorted to by the Acting Government Medical Officer of Health among the villages on this coast. While in 1917 the East Coast furnished 119 out of a total of 698 cases, from January to December, 1918, this group furnished but 28. Other districts shewing reduction have been the West Bank of the Demerara River, from 26 cases in 1917 to 5 cases in 1918 (to October, 31st), the West Coast district from 57 to 32 up to October 31st, 1918, and certain areas in Berbice County.

On the contrary, Georgetown has been responsible for no less than 206, and the East Bank group 160 cases to the same date.

These groups and the Suddie, Essequibo, district are the only ones to shew an increase or a slight decrease.

The following table shews the approximate population of ten centres in the colony, the total number of people inoculated in 1917 and 1918, the percentage of the population inoculated, and the number of notified enteric cases, in the latter case from 1916 to 1918. The inoculations were carried out chiefly in 1917 and 1918, though in the case of the East Coast and the East Bank districts, some were also done in 1916:—

District.	Population.	Total No. Inoc'd.	Percentage of Persons Inoc'd.	Notifications of Enteric. 1916.	1917.	1918 (to Oct. 31).
1. Georgetown	54,000	1,431	2·6	190	272	206
2. Buxton (East Coast) ...	26,000	3,107	12·0	34	67	8
3. Plaisance (East Coast) ...	23,000	2,396	10·4	24	52	20
4. West Coast	30,000	1,200	·4	18	57	32
5. West Bank	13,000	1,227	9·4	8	26	5
6. Berbice County	60,000	824	1·4	24	72	32
7. New Amsterdam	8,000	225	2·8	2	16	8
8. Essequibo	22,000	770	3·5	7	19	41
9. Wakenaam Island	5,000	170	3·4	4	7	1
10. East Bank	17,000	4,038	23·8	51	110	160

It will be seen that those centres in which a number in the neighbourhood of 10 per cent. of the population has been protected shew a distinct decrease in the number of notifications, with the single exception of the group of estates and villages on the East Bank. Several different explanations of this are possible.

For example, it has been necessary to group together both estates and villages on the East Bank, whereas nearly all, or, at any rate, the vast majority of the inoculations were done on certain estates.

Secondly, the figure given for the number of inoculations has been based on the number of vaccines sent out from the laboratory, but, whereas unused vaccines are returned from some districts, it was discovered that those unused had been kept on the estates in some cases, including those at the Peter's Hall group of estates.

In the case of this group, I have not been able to ascertain exactly how many doses were actually used. The numbers issued, as will be seen, were very large, and it is possible that a large percentage of these was really unused.

With regard to the efficacy of the vaccine, records have unfortunately not been kept with regard to cases and deaths occurring in the inoculated, except in cases inoculated by

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me, but opinion is unanimous that the rate of incidence and mortality among the inoculated is considerably lower than among the unprotected.

Other explanations of the increase in the East Bank district are available, but do not affect the conclusion which, it appears to me, is to be drawn from the facts given above.

This conclusion is that inoculation on a voluntary basis—and compulsion appears to me undesirable, and, perhaps, impossible—is not to be depended on as a method of preventing the spread of enteric in civilian communities in the tropics, unless a number in the neighbourhood of 10 per cent. of the population submits to inoculation.

It would be hardly possible without very energetic efforts on the part of the Public Health Staff to get a much larger proportion to submit voluntarily to inoculation except during an epidemic, and it will, therefore, be apparent that other measures, such as a vigorous sanitary propaganda and the instalment of an efficient system of sewage disposal, are at least as necessary as inoculation.

Granted these, however (and the indications are that the city will shortly be in possession of a scheme for disposing efficiently of its sewage), inoculation is of real assistance in still further reducing the incidence of the disease.

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TRANSACTIONS

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SOME CONSIDERATIONS REGARDING PREVENTABLE DISEASES AND THEIR PREVENTION.

Presidential Address

BY PROFESSOR W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P.

My first duty is to thank you for the great honour you have conferred on me in electing me your President. Proud as I am of holding this honourable position, I confess that I feel some trepidation in succeeding to an office which has been held by men so distinguished in different branches of tropical medicine and research. All my predecessors have been eminent in their profession, and MANSON, ROSS, BRUCE and LEISHMAN have, in addition, been leaders of the highest renown in the advancement of medical science. The discoveries in filariasis, malaria, n'gana, human trypanosomiasis and kala-azar have unfolded the wonderful life-history of the parasites of these diseases, demonstrated the methods by which they spread, and opened up quite a new field of research, which has been eagerly explored by a large number of enthusiastic and brilliant workers. The result is that already our knowledge concerning the intimate connection between many of the communicable diseases of man and beast and protozoa, helminths and insects has been greatly increased.

If I were to chronicle the knowledge acquired within recent years in this direction, it would involve, for the most part, a summary of the work done by the Fellows of the London Society of Tropical Medicine and Hygiene at home and abroad. The establishment of the Schools of Tropical Medicine in London and Liverpool and their teaching, have infused a new spirit into the Tropics for research and advancement of preventive

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medicine, and much valuable and important work, dependent on individual effort, has been accomplished by the initiative of former students of the respective Schools.

The Value and Need of Different Lines of Research in Preventive Medicine.

The investigations into the natural history of the animal parasites, revealing the intimate and active relationship of the insect world to the causation and dissemination of certain tropical diseases in man and the lower animals, were of a different kind to those pursued before, and mark a new era in medical research. Their success represents a further advance on the road to that fuller understanding of natural phenomena which were a mystery to our forefathers, and many of which are still a mystery to us. There is already ample evidence of the great power which the exact knowledge acquired by this line of research, when applied, places in the hands of the sanitary administration. There is the successful work done by Ross at Ismailia, in the Mauritius, Cyprus and Greece; by Watson in the highlands and lowlands of the Malay States; by Gorgas in Havana and the Panama Canal; and by the Sanitary Department on the West Coast of Africa. The search for the breeding-places of the larvæ of disease and the methods introduced for their abolition is a new feature in modern sanitation. The results already obtained bid fair to compete with those of the bacteriological or botanical era which preceded it, and which by its revelation of the causal agents of a large number of infectious diseases linked up man's relationship not only with the vegetable world but also with that of the lower animals. The two have shewn us once more, from different standpoints, how intimate is man's relationship with his environment, and how, even under the most difficult conditions, by the application of that knowledge, protection and safety can be secured. The continuous and systematic application of this knowledge to civil populations in the Tropics is what is now required. The real trouble is that the knowledge we possess is only spasmodically and very restrictedly put into practice. We are familiar with the results of the practical application of bacteriology in epidemic disease in man and the lower animals, both in its detection and prevention. The latest demonstration of its power, when applied on a large scale, is the remarkable freedom of our armies in the West from typhoid fever, and in the East from cholera and plague. Inoculations against these diseases, against tetanus and wound infections, have removed what were formerly great sources of illness and mortality.

In growing bacteria in our laboratories, studying their characteristics, their increase and decrease of virulence, the effect of humidity, dryness, electricity and other agents on them, and in experimenting with them, we obtain a faint glimmer of the happenings of these micro-organisms in the great laboratory of nature.

Some day, perhaps, as science advances, our descendants will be able to understand the causes of the periodic appearances and disappearances of those great epidemics and pandemics due to bacteria and, perhaps, also those due to animal parasites. Then they will also be able to explain, apart from the greater supply of food, the causes of the periodic multiplication, swarming and migration of locusts from their endemic homes, and their subsequent limitation to the area from which they came; also of the plagues of caterpillars and agricultural pests, and a host of other puzzling phenomena.

Our ancestors attributed outbreaks of disease to an epidemic constitution of the air and its influence on man's economy. That epidemic constitution has still to be studied. It has been lost sight of in recent years, though earlier it engaged the attention of philosophers, who ascribed the origin and exceptional spread of epidemics to unusual meteorological conditions which caused floods, drought and famine. Who shall prove that they were wrong? What effect have floods, drought and famine on the parasites of disease, or on the various animal and vegetable micro-organisms that teem in the soil? Do the parasites gain virulence and increased powers of diffusion in the soil, or in the diseased vegetable world subjected to these conditions, or in the herbivorous animals that subsist on it, or in the vermin and insects that feed on the diseased animals or plants or in man, who, when famished, is glad to eat any vegetable, seed, grass, fruit or meat even if diseased?

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Apart from the virulence and diffusiveness gained by the sojourn of the microbe in one or several of these media, what are the factors which combine to form an endemic area for a predominant microbe? The geological nature of the soil often limits the geographical distribution and kind of animals living underground, and probably the kind of insects and micro-organisms. Is it in this direction that the explanation may be found concerning the endemic homes of cholera, plague, influenza, yellow fever, bilharzia and cerebro-spinal disease? Was CUNNINGHAM, of Calcutta, on the right track when investigating the nature and composition of the soil, and its gaseous, organic and inorganic contents? Is it the interaction of unusual meteorological conditions on the local fauna and flora of certain limited areas that prepares the micro-organisms for their migration beyond their endemic homes; not by the air, as BRYDEN and others thought of cholera, but by making use of man, the lower animals and insects, and sometimes inanimate objects, as carriers? As regards sudden predominance of a particular microbe, I remember once in Calcutta a pond or tank which contained in its water the ordinary microbes found in others. Suddenly it developed only one kind, the remainder having disappeared. What produced it I was unable at the time to determine. Was it some slight alteration in the organic or inorganic constituents in the water that favoured the growth of a single organism at the expense of the others? It appeared to me, if this could occur with one kind of microbe it might do so with another, such as the cholera bacillus; and if in the water, why not in the soil, where, on the surface of earth drains, the cholera bacillus was found?

INVESTIGATIONS ARE NEEDED IN THE ENDEMIC CENTRES.

There must be explanation for all these phenomena if we could only lift the veil.

It is necessary to know more about the endemic areas of disease. They should be sought out and classified. It is there that the natural history of the disease in man can best be studied in relation to the geology, fauna, flora and other conditions existing in the locality, and it is there that the conditions have to be investigated, to give us a clue to the factors which change a sporadic or endemic non-spreading or self-limiting disease into a diffusive and wide-spreading epidemic or pandemic. This knowledge will also allow us to adopt successful measures at the fountain-head.

Although much has been learnt regarding preventable disease, there are still vast problems awaiting solution by research in preventive medicine. We require to know more about plague, cholera, small-pox, leprosy, influenza, cerebro-spinal fever, trypanosomiasis, yellow fever, bilharziasis, blackwater fever, and many other tropical diseases, and their relation to man's environment, not only in his home but also in nature. Where these diseases most commonly prevail research laboratories are necessary, well equipped with all the modern resources which bacteriology, protozoology, parasitology, biochemistry and geology place at our disposal. The investigations need to be on a comprehensive scale, and not, as I found in Uganda in 1913, where Miss ROBERTSON and Mr. FISKE were the only representatives investigating trypanosomiasis in a part of the country that, in ten years, had been denuded of nearly a quarter of a million of its inhabitants from this disease and where rinderpest was attacking the cattle.

THE CONDITIONS WHICH CAUSE DISSEMINATION IN TIMES OF PEACE AND WAR.

Once an endemic disease has acquired diffusive qualities there are ample means for its transport and dissemination. Commerce, movements of labour, emigration, pilgrimages in times of peace and the movement of armies and refugees in times of war favour transport, while the insanitary conditions of the locality favour dissemination. The conditions to be found in most countries, even in the twentieth century, are highly insanitary, and even in the best, as in England, there are plenty of slums. The epidemic will find in the huts and houses of the peasants, labourers and poorer classes the fostering conditions for its invasion and spread. If the disease is plague or typhus, the conditions

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are ideal where the family live in the same dark and ill-ventilated room or house with the donkey, pig, ox, poultry, rat and insects of each.

If the disease is cholera the exceptions are few, especially in the Tropics, where a polluted water supply, a primitive disposal of sewage, much filth, numerous flies, and overcrowding are not available conditions for its spread.

In times of war these and other conditions become accentuated by deficiency of food. We realise to-day, better than we have ever done before, what war means to the inhabitants of an invaded country. In previous wars on the Continent, as in the Great War of 1914-1918, it meant the loss to them of their crops, more complete than that sustained by the worst swarm of locusts, the seizure of their cattle and other live stock, the destruction or occupation of their homes, and then hunger, sickness, misery, hardships and the aggravation of insanitary conditions, accompanied with or followed by pestilence. The longer the war lasted the more complete the effects. The population of Germany in the Thirty Years' War was reduced from 17 millions to four, worn down by fighting, famine, and pestilence. The world war of 1914-1918, though shorter than many other wars, was on a more gigantic scale, and instead of towns being besieged as formerly, whole countries were blockaded by armies and fleets. Not only did invaded countries suffer, but also those that were blockaded. Epidemics, varying in kind, broke out in both, but in the armies of the West the trained sanitary organisation and adequate food supply were so complete as to immediately check and prevent any considerable extension either in the army or among the civil population over which the military had control. It is an object lesson of importance as to what can be done by good administration. It is also in striking contrast to the failure, in the early years of the war, in the prevention of tropical diseases in Gallipoli, Mesopotamia, East Africa, Egypt and Salonika, due to the causes explained in my paper on the *Sanitary Aspects of Warfare in South-Eastern Europe*, read before this Society in October last. In this connection it is only fair to state that great credit is due to the Mediterranean Sanitary Committee, whose work did so much to mitigate and remove the conditions which they found inimicable to the health of the troops, and which a short-sighted policy had permitted to arise. There were two diseases which were not checked in the West, and for the same reason in both, viz., insufficient knowledge. One was the great prevalence of trench fever, whose transmission of infection by the louse was not established until late in the war; and the pandemic of influenza, the origin of which is unknown, but which in its intensity and destructiveness throughout the world might, in a slighter degree, be compared with the havoc of the pandemic of pneumonic or influenzal plague of 1348, in the time of the Tartar Wars.

The Aftermath of Great Epidemics on the Nervous System.

We have much to learn about influenza. It has passed over the whole world, and we are not much wiser as to the protection of communities against it. Has it an endemic home or homes like cholera and plague, and where is it? The pandemic appears to have left its mark. Is it a mere coincidence that the pandemic of the fourteenth century, and that of the twentieth have been followed by manifestations of mental disorders, hysteria and unbalanced judgments? In some countries the disturbances have been worse than others, evidently affecting more acutely that proportion of the population whose nervous system is more unstable. It is a matter of interest in this connection that nervous and mental symptoms were observed in the epidemic of 1890, and that Clouston held the view that the great epidemic of 1890, which was mild compared with that of 1918, had left the nervous and mental tone of Europe and America lower by many degrees than it found it. Judging by the profound effect caused by the spirochæte of syphilis, on certain subjects, Clouston's view is one that needs to be carefully considered. If certain epidemics, in addition to their already known physical effects, result in a lowering of the nervous tone and conduct among even a limited number attacked, it is an additional reason for their prevention. This aspect of the question, if confirmed, enlarges the sphere of preventive medicine and emphasises the importance of its practical application.

The immediate effect of war and the epidemic of influenza are to be seen in the

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disorganised state of the greater part of Europe at the present time, a year after the war, and the prevalence of epidemics. Cholera and plague are epidemic in Russia. Typhus fever and relapsing fever are spreading and destroying large numbers of the inhabitants in Poland and the adjacent countries, and small-pox is gradually spreading over the continent. Except in regard to typhus fever, which is a disease more of cold countries than the tropics, we can hardly expect that our tropical possessions will escape from an exacerbation of the preventable diseases which prevail in them under normal conditions.

While recognising there is much to learn regarding the epidemiology of many communicable diseases and the causes of industrial sickness, yet it must be recognised that we have sufficient data for the prevention and suppression of the most common and most destructive diseases in the tropics if we will only apply that knowledge.

War and Epidemics have compelled the application of Sanitary Measures based on the knowledge already acquired.

War and epidemics bring out the weak spots in our armour of defence against disease, but their after-effect is generally to set men's minds actively in train to devise means for future protection, and ideas and schemes that have for many years only created a languid interest suddenly get energised into activity and become a reality.

This is seen to-day in the creation of a Ministry of Health in this country which has been advocated for fifty years, but which the war and epidemic of influenza has quickly brought into being. It is seen also in the determination to grapple with the housing question and the problem of the "C3" men, women and children in the population.

It was the Boer War, with its enormous number of dysentery and typhoid fever cases, that led to the reinstatement of the Sanitary Officer in the Army. There had been a reactionary movement for some ten years before the Boer War, gradually undoing the efficiency of the organisation of sanitary officers which had been the outcome of the Sanitary Commission reporting on the Crimean War. For nearly that period no sanitary officer was appointed at Aldershot, a position formerly held by a Deputy Surgeon-General. The first-fruits of this policy were the large number of officers and men who lost their lives from preventable disease in the Soudan Campaign.

The Navy led the way in protecting her fleets against disease in the tropics. During the wars with France and Spain in 1778, and following years, the West Indies was the principal seat of naval operations, and much greater fleets were then employed in that quarter of the world than in any former period. There was much sickness in the fleet, varying greatly in different ships, and consisting of dysentery, scurvy and fever. Dr. Blane (afterwards Sir Gilbert Blane), when physician to the fleet under Lord Rodney, had monthly returns sent to the headquarters of the fleet recording the sickness and deaths in each ship, and anything else that related to the health of the ship. He found that the average mortality from disease in twelve months was 1 in 7 of the seamen, and on an average one man in 15 was on the sick list. In 1781 he addressed a memorial to the Board of Admiralty, pointing out the excessive mortality, and expressing his conviction that if certain proposals which he suggested were carried out, more than two-thirds of the seamen who died in that climate would be saved.

These recommendations were similar to those proposed by Dr. James Lind, a naval surgeon, in 1757, in his essay on the most effectual means of preserving the health of seamen in the Royal Navy, containing cautions necessary for those who reside or visit unhealthy situations, etc. Sir Gilbert Blane early recognised the fact that little improvement could be effected in the health of the fleet when left to the initiative of individual officers, and that sanitary rules and regulations, based on scientific knowledge, must be issued by the Admiralty for them to be carried out systematically and efficiently. His recommendations were first put into practice in the West India Fleet with excellent results, and later they were extended to the whole of the Navy. The improvement of the health of the Navy and the reduction in its death-rate contributed to the victories in the revolutionary war. Sir Gilbert states at a later period, "that if some new means of

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preserving the health and lives of seamen had not been discovered and put into practice, the whole stock of them must have been expended before the conclusion of the revolutionary war—that is, between the years 1793-1815."

The improvement has continued, notwithstanding the changes in the warships, and instead of the average mortality in twelve months being 1 in 7, it is now 1 in 310.

We know what the Navy has done in the Great War of 1914-1918.

We are apt to forget the lessons of the past. For instance, in the Mesopotamia campaign there would have been no scurvy if the history of the Navy in relation to that disease and its abolition in the latter part of the eighteenth century had been borne in mind and acted upon. It should not have required the valuable and admirable work of Miss HUME and others of the Lister Institute to resuscitate the necessity of the supply of vitamines in the food stuffs supplied to the Army.

One is apt to forget that the use of cinchona bark as a prophylactic goes back to at least the early part of the eighteenth century. Dr. JAMES LIND, the pioneer of sanitary reforms in the Navy more than 160 years ago, mentions that the continuous use of bark is an effective preservative against a relapse of malarial fever, and that "on the coast of Guinea the factories were furnished with proper quantities of the bark by the late African Company, which was taken by way of precaution during the rainy and sickly season, and it was attended by remarkable success with such as could be brought to a regular course of life, and refrain from eating such quantities of animal food as they were wont to do in England, which yearly destroyed many on that coast (Guinea)."

One sometimes wonders whether the many relapses of the soldiers from Salonika and other seats of war were due to our preference for the alkaloids of quinine, for which there has been an unprecedented demand, and some of which may have in their preparation lost the complete qualities of the cinchona bark, which itself varies in quality. Are we quite sure that the procedure which destroys the vitamines of canned food may not affect some of the properties of the alkaloids?

The Crimean War, with its tragedies, owing to want of organisation, sanitary and otherwise, and the success which followed the work of the Sanitary Commission, of which Miss NIGHTINGALE was an important member, led to important sanitary reforms in the Army at home, and in 1859 to the appointment of a Royal Commission to enquire into the health of the Army in India, which in its report published in 1863, disclosed a deplorable condition of things and a death-rate of 69 per 1,000, although in the later years of the Honourable East India Company, the conditions in regard to health and the comfort of the soldier had been materially improved, and the mortality had been reduced from 80 to 69. In consequence of this report, remedial measures were at once applied to the Army in India, and were extended to the towns and districts adjacent to the cantonments.

THE BEGINNINGS AND PROGRESS OF A SANITARY ORGANISATION IN INDIA.

The Royal Sanitary Commission were convinced that if sanitary measures were applied to India under an organised Public Health Service, the health of the people of India would in time reach a high standard. It was recommended that such an organisation should be created both in urban and rural areas in India. In 1864 a Sanitary Commissioner was appointed for each Province in India for the purpose of improving the sanitary conditions of the people. The Civil Surgeon, ordinarily during peace an officer of the Indian Medical Service, temporarily placed at the disposal of a Local Government, was empowered to advise the Civil Commissioner of the district on sanitary questions and later to act as sanitary adviser to municipalities newly formed, for the purposes of local self-government. Gaols were also placed under medical supervision and special health officers were appointed for Calcutta, Bombay and Madras. Later, a very important addition was made by the establishment of Central Boards of Health in each province, having for their function the criticism in professional and financial aspects of major schemes of water supply and drainage prior to their sanction by Local Governments. The work which the organisation here described has been able to accomplish is very

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creditable, considering that a province in India is sometimes about the size of France, and a civil surgeon's district in which he practises is often as large as a third of Scotland. Vaccination, introduced as a protective measure in 1802, was stimulated; then followed the introduction of conservancy, drainage and water supply into some of the more important towns; the registration of deaths, the compilation of statistics, and the regulation of large fairs and pilgrimages, which experience shewed to be always dangerous centres of disease.

The effect of these and subsequent health measures has been to reduce the death-rate of British troops from 69 to a pre-war rate (average 1910 to 1914) of 4·51; of Indian troops from 20 to 4·39; and of prisoners from 82·7 to 19·19 per 1,000. Notwithstanding these successes it is obvious that an organisation which consists of a Sanitary Commissioner, Sanitary Engineer and Sanitary Board for each Province, even with the assistance of the Civil Surgeons, is quite unequal to the task of dealing with the sanitary administrations, both rural and urban. It must be remembered that Civil Surgeons of Districts, or, as they are called in the Madras Presidency, "District Medical and Sanitary Officers" —who ordinarily, during peace, are officers lent temporarily by the Military Department to the Local Government—are expected to attend to the medico-legal work of the district, the medical necessities of civil officials, perform the executive medical and surgical work of all local hospitals and institutions including gaols, and hold administrative and inspecting charge of numerous hospitals and dispensaries scattered throughout their districts. In illustration of the extent of work they are expected to fulfil, I may state that at the Indian Medical Congress of 1894, Dr. HART, then editor of the *British Medical Journal*, made a great point of the absence of research results from the India of that date—there then being only one laboratory specially equipped for this purpose. He had, obviously, fancied that, having regard to the rich possibilities afforded by India, the Indian Medical Service men were not workers. After the Congress he was the guest of several Civil Surgeons before his departure to England. From the experience thus gained, in a speech given at Madras, he drew a contrast of the working-hours of busy men of the various professions in England and the Indian Medical Service officers in civil employ, and no longer expressing astonishment at the absence of research, he declared the life was so strenuous as to be a "dog's life."

BEFORE INDIA WAS INVADED, OR EVEN THREATENED, BY THE CHINESE PLAGUE, THE NEED OF A MORE COMPLETE SANITARY ORGANISATION WAS RECOGNISED.

It is also certain that the organisation was quite incapable of dealing with an epidemic, even though a Sanitary Commissioner was appointed by the Government of India. The defenceless state of the country invited attack from any epidemic disease, indigenous or imported into the country. It was these considerations that induced me to bring forward in 1894, at the Indian Medical Congress, a scheme for the establishment of special sanitary service for the civil population of India, which was accepted by the then Local Governments and the Government of India in 1895, but has not even now been brought into operation. The most advanced sanitary administration at that time was that presided over by Colonel KING, C.I.E., as Sanitary Commissioner of the Madras Presidency, in which there were a District Sanitary Officer and Assistants. Here, in 1881, on the recommendation of the then Sanitary Commissioner (CORNISH), there was attached to each District Medical and Sanitary Officer an Assistant-Surgeon, capable of taking charge of the Headquarter Station during the absence of that officer on sanitary duty in his district. To this then existing staff, in 1893, Colonel KING advised the compulsory addition of technically trained Sanitary Inspectors, qualified by examination. The scheme was sanctioned in 1894, and the men now number over 600. But his efforts to secure permanent appointments for these beyond municipal areas, although approved by the Madras Government and successful in some districts, was largely inhibited by local authorities. The surplus men now form a reserve for epidemic duties, on the old principle of locking the door after the horse is stolen, and many find after further technical

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training *ad hoc* appointments as Deputy Inspectors of Vaccination. By later elaboration of staff (1904) there was secured sanction by him from that Government for a second Assistant-Surgeon attached to each District Medical and Sanitary Officer, intended solely for sanitary duties in the District, as well as a Public Works Overseer, capable of making plans and estimates to aid the District Medical and Sanitary Officer in formulating recommendations for minor sanitary and anti-malarial works; and further advances were made later, but owing to the opposition of local authorities of districts, even in this comparatively advanced area of India, there is failure to reach the reasonable ideal, suggested by me at the Congress of 1894, namely, that a specially trained and separate sanitary service should be arranged for, quite independent of physicians in connection with the hospital or of those in private practice; that there should be schools of hygiene and preventive medicine and also of sanitary engineering, and that diplomas should be granted to those who were trained in these schools; and, fourthly, that laboratories for research should be established in various parts of India. Colonel KING, C.I.E., in a remarkably able and statesmanlike address before the East India Association, in July, 1914, points out that, notwithstanding the advice of the Royal Commission, conveyed in the Report of 1863, the urging of Provincial Sanitary Commissions, successive Sanitary Commissioners with Local Governments, the first Indian Medical Congress and the Royal College of Physicians in London, the proposed organised efforts of 1914 were confined to municipalities, which only represented the provision of a sanitary service for the protection of 10 per cent. of the population. It may be asked, What is to become of the other 90 per cent.? As shewn by Colonel KING, the death-rate in India varies in sixty-four towns from 30 to 70 per mille, and the expectation of the young Indian at birth is 23½ years less than that of an English child. He mentions that there are annually one million deaths from malaria, and over a quarter of a million deaths from cholera, that small-pox destroys thousands of lives annually, and that there were in the sixteen years since the plague began in 1896 some eight million deaths from that disease. This number has since increased to ten millions. No one can be satisfied with this condition of affairs. The scheme is to have 25 Sanitary Commissioners, 12 Deputy Sanitary Commissioners, 34 first-class Medical Officers of Health, and 104 second-class Medical Officers of Health for a country which, excluding Russia, is the size of Europe, and has a population under direct British rule of over 244 millions, and a total population of over 315 millions. The area of British India is 1,097,901 square miles against 58,338 of England and Wales. Compare these figures with England and Wales, which, some years ago, had 1,700 local Medical Officers of Health, 40 County Medical Officers, and 26 Medical Inspectors, besides the Inspectors attached to the Board of Education and the Home Office, and which now has an additional staff under the new Ministry of Health. The War cut short even this diminutive scheme of the Indian Government, and the last announcement is that it will be put into operation as soon as possible.

One cannot recognise this as an adequate service for India, and I am convinced that until a proper sanitary service for India is formed—which would be a splendid outlet for young Indian medical men and women—there will be no real progress, and India will remain defenceless against epidemics. The root of the matter is there is no Minister of Health in India, whose duty is concerned solely with sanitation and the health of the population. Public health should not be, as it is in India, a by-wash of the Educational Department, in which it is placed as a sort of miscellaneous item along with archæology, and in which the money goes to Education, and Public Health is starved, and in which the topsy-turvy doctrine is held that the people must be educated before they can be protected against disease. No country has waited until its people are educated before it introduced sanitary reforms, and least of all England. Major JUSTICE, the Sanitary Commissioner with the Government of Madras, in his official report for 1911, states that "to wait till the people are educated will be to indefinitely postpone the evil," and Dr. TURNER, the late Health Officer of Bombay, remarks that the millennium will have arrived when the people voluntarily carry out sanitary matters. The only education in sanitary matters the people understand is sanitary laws and by-laws, and this is the form education should take. It was the form which the wise men in ancient times employed

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when they included sanitary laws in their sacred code. As quoted by Colonel KING, the Education Minister thus defines the position: "Our first and signal objective is to educate the people as to the value and the necessity of measures for protecting them in their homes and their lives and those dearest to them from the ravages of plague and malaria, cholera and other communicable diseases, and all the miseries which follow in their train. Fortified by the results of research in India, we can leave the future with confidence to preventive medicine and preventive sanitation." When a question was asked in the Bengal Legislative Council what this implied, the reply was that protected water supplies were to be arranged for the people *when they have been taught to appreciate them.* It is difficult to have any patience with such an attitude of mind, and it is no matter of surprise that, with such views, the troops in Mesopotamia were not furnished with the ordinary requisites and organisation to protect them from the ravages of disease and all the miseries which followed in their train. They were left to be educated.

The mode in which cholera spreads and the methods by which it can be prevented are well known. Yet in the North-West Frontier there were no precautions taken until the outbreak of this disease in the Afghan campaign of 1919 alarmed even the Government of India. It was not then a matter of education but a tragedy, and public opinion forcing the Government to do its duty, and institute measures to protect the troops and camp followers, which it had neglected when it substituted fine phrases about waiting until the people were educated to appreciate the benefits.

Research can be made an excuse for doing nothing. My views concerning this in regard to India were expressed some years ago in the following terms: "We hear a good deal about research, and research is much cheaper than the *application* of sanitary matters. No one has a greater respect for research than I have. A few years ago, I urged that sanitary research and administration should go hand in hand. That is the real position. At the present time a good deal of research has been done—very useful in its way—and, no doubt, it will be fruitful of results, but I consider that the application of sanitary measures, on the basis of what we already know, is the urgent need of India. I will give you an illustration of this. Supposing the Government of India had been asked to construct the Panama Canal, the kind of proceeding they would have introduced with the present *régime* would have been to send out men to make researches; they would not have based their actions on the discoveries already known of ROSS and FINLAY as the Americans have done. These investigators would have found that the mosquitoes there were a little different from what they were in other places where malaria and yellow fever existed, and, under the circumstances, the recommendations would have been made to postpone the matter until a further discovery was made. I am convinced that sanitation will not receive adequate attention in India until there is a Ministry of Health, whose duty is concerned solely with sanitation and the health of the population" Then will the organisation required for urban and rural sanitation be formed, and India will at last be able to throw off the incubus of plague and cholera, and protect itself against other preventable diseases.

The Egyptian campaign led to the formation of a Civil Sanitary Service in Egypt, which did some excellent work, but which is, as shewn by the Commission of which Colonel BALFOUR was Chairman, inadequate for the public health of the country. The reorganisation which this Commission recommends covers what a Public Health Service should be under a Ministry of Health It is a most complete scheme, and one worthy to be followed in other countries, modified, of course, according to local requirements. I am glad to see that the United States of South Africa are to have a Minister of Public Health and a General Public Health Act. It is, indeed, a great advance.

THE EFFECT OF CHOLERA EPIDEMICS IN ENGLAND.

For the civil population of England, it was the cholera epidemic of 1831 that brought about the various Sanitary Commissions which revealed the very insanitary condition of the towns, with their cellar dwellings, bad housing, slums, and overcrowded and filthy

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courts, under which the rural inhabitants were living who had migrated into the towns owing to the new industrial period. It was the threatening of the epidemic of 1849, rendered famous by SNOW'S discovery, thirty-five years before the cholera bacillus was found, that the disease was spread by water contaminated with excreta from a cholera patient, that led to the establishment of a Central Board of Health with sanitary powers, followed by local sanitary administrations and a Public Health Act, which, together with the series of investigations carried out by Sir JOHN SIMON, BUCHANAN, and the other distinguished men of the Privy Council and Local Government Board, have changed the whole aspect of the country into a clean and comparatively healthy one. SNOW'S discovery ultimately, but only very slowly, swept away the time-honoured view—and one which was held in great favour by the Indian Government for forty years afterwards—that the disease was air borne. It was as efficient in this respect as the discovery of ROSS, which dissipated the theory that malaria was due to miasma from marshes. For those men in the Tropics who are away from laboratories, and have no microscopes, there is comfort and encouragement that SNOW'S discovery and the knowledge acquired by subsequent investigators as to the vehicles by which diphtheria, scarlet fever and typhoid fever were spread, were ascertained without these modern resources; and, further, that it has been essentially by applying systematically and continuously the knowledge thus acquired to remove the fostering conditions favouring the prevalence of preventable disease that improvements in public health have been and can be effected.

Though, compared with the Early Victorian period, there has been an immense improvement in the health and sanitary condition of the population, the problem in England has not been wholly solved, as shewn by Sir GEORGE NEWMAN'S recent memorandum and the disclosures regarding the housing conditions.

It is over thirty years since the Royal Commission on the Housing of the Working-Classes sat, and yet until the other day, owing to the absence of a Central Authority, with broader views than have prevailed, and with greater initiative and powers, although millions have been spent on demolition of slums, much of the good work has been nullified by unhealthy houses being allowed to be built, forming other unhealthy areas. We have now, at last, a Ministry of Health. But to abolish slums, and to prevent new slums arising, a much stronger Act than that which has recently come into force will be needed. To accomplish this, town planning should be compulsory, and building acts must have greater control over the provision of light and air for houses and their rooms.

The same vested interests and ignorance of what is necessary for a healthy community, which obstructed and prevented Sir Christopher Wren, who was the first British town planner, carrying out his scheme of making London the most beautiful and healthy city in the world, have marred with ugly and unhealthy spots the cities, towns, and even villages of England.

THE HOUSING QUESTION IN THE TROPICS.

The same problem, even in a more acute form, exists in our tropical possessions, which are about nine-tenths of the Tropics. I am not referring to the old towns that existed in these countries before we went there, but to the towns that have grown up under our protection. Having taken an active part in the demolition, under Cross's Act, of a very insanitary area, the haunt of typhus fever, in Aberdeen, and having given evidence before the Royal Commission on the Housing of the Working-Classes, I was particularly struck, on arrival in Calcutta, by the contrast between the airiness of the European quarter and the crowding together of houses on very limited space which distinguished the native quarter. It was very picturesque but exceedingly unhealthy, and I determined to secure a Building Act for the city. It took ten years before a Commission of Enquiry was appointed, but I had the satisfaction of the Commission taking a broad view of the importance of good housing to the health of the family and the community. The result was a Building Act which formed a model for the other towns in India and the Far East. The plague soon sought out the rookeries in Bombay; it did the

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same in Hong Kong. In both it entered the dark, ill-ventilated and overcrowded tenements and attacked the inhabitants. In visiting these places, it sometimes needed a lighted match or lamp to examine the occupants.

Sir Stamford Raffles—one of the many great Englishmen who have done such magnificent service for their country—laid out Singapore on broad and sanitary lines, but those who came after him did not realise the evil that would arise by allowing the Chinaman to cover the whole of his building plot with masonry dwellings. Some of the blocks shew tuberculosis almost epidemic, and this in a locality where cows are free of tuberculosis, owing to their being housed in open sheds. A Commission has just issued a report in which there are certain recommendations of demolition, etc., all of which involve a very large expenditure. The Malay States have taken the lesson to heart, and, in their smaller towns, regulate their buildings so that only a small part of the plot shall be covered. In West Africa, the same conditions, giving rise to unhealthy areas, were arising in the town and trade centres, but the sanitary services, which were established there ten years ago, and which have done splendid work in improving the health conditions in each colony, have fully recognised the evils, and have taken measures for prevention rather than amelioration. Town planning and building regulations are the forces they are bringing to bear on the question.

In East Africa and Uganda, comparatively new countries, Mombasa is the only town that is old; the others and the trade centres are all new. Yet the same mistake of neglecting to plan them on sanitary lines, and to regulate the buildings as to space and healthy requirements, has continued. The Indian Bazaar in Nairobi, and the insanitary conditions in other parts of the town, are not only a danger to the town and the whole country, but also a discredit to all concerned. The result has been that, with African and Asiatic living under conditions which are extremely unhealthy, and where an organised sanitary service is only about to be introduced, we have these towns and trade centres constantly affected with plague, small-pox, dysentery, malaria and other epidemic diseases. The men are also being weakened by the large percentage suffering from ankylostomiasis; 77 per cent. of labourers from Mombasa, and 80 per cent. of those admitted to the Nairobi Hospital, have been found to be suffering from this disease. The losses from preventable disease during the last few years have been exceedingly great. There cannot be the slightest doubt that some of these epidemic diseases are capable of becoming even more formidable and destructive as long as existing conditions are permitted to remain, and as long as there is no effective sanitary organisation supplied with the requisite funds and powers to take the necessary measures to remove these conditions, to check and prevent disease, and to preserve the native population, which is comparatively small, and which is the chief asset of the country.

A Wider Outlook from a Health Point of View is required in the Tropics.

Our outlook in regard to native populations must be wider and more sympathetic from a health point of view than it hitherto has been. For instance, in East Africa, speculation in land raising the price of house-plots to a ridiculous price in a country which is four times as large as England and Wales, and with a population ten times smaller, must not prevent the local Government from seeing that adequate building regulations are enforced, that town-planning shall be introduced, even if it be unpopular and at a loss to the speculators; and seeing also that African and Asiatic shall be housed in their respective quarters in a decent and healthy manner; that their houses or huts shall not be crowded together in the wide expanse of the plain, as if land were gold and not available; that each house or hut shall have a good plot of fenced land attached to it for domestic use; and that there shall be parks and open spaces in their own quarters, in which the children and adults can have opportunity of recreation and rest, and that the towns and trade centres that are now in the making shall grow up beautiful and healthy.

Our responsibilities are great, and it is essential that we should carry them out in a large and generous spirit, and not with that favourite and uneconomical excuse that

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there are no funds available for the preservation of the health of the people. The application of sanitary science is not expensive in new countries; on the other hand, it is its neglect and the ultimate necessity of remedying the defects that are costly.

From a very wide experience in different parts of the world, I am convinced that, notwithstanding the progress in certain directions in sanitation, much of the ill-health and sanitary difficulties in our Crown Colonies and Dependencies is due to our feeble, short-sighted and haphazard policy in the development of our towns, and the absence of any special attention to building regulations and the prevention of subdivision of plots and of houses. I consider that, next to food and water, the fundamental basis on which a community depends for health is the housing question. The first step in that direction and in civilisation is the separation of the lower animals from the family's living apartments or house. The separation should be complete, and the animals quartered well away from the house. The next is that the house and the rooms in it shall have abundance of light and fresh air, and that the house-plot shall have its own ample-fenced and uncovered space for domestic purposes and for the children. The third is that the houses shall not be jumbled together as if they had been thrown down like a cartload of bricks, and spread over the ground without order.

In the Tropics one has seen places where it would be impossible to introduce sewerage, and places where sewerage, when introduced, was anything but a health-producing agent. It goes without saying that the waste products of the inhabitants shall be removed, that there shall be a protected water supply, that the breeding-places of mosquitoes shall be abolished, and that preventable diseases shall be prevented. But none of these things can be done without sufficient sanitary powers embodied in a Public Health Act, and an adequate and organised sanitary service, central and local. Except in the case of water supplies, conservancy, vaccination, and attempts to check epidemics when they are well advanced, all of which have been for the common benefit of the community, native and European alike, it is exceptional that great efforts have been made in the intervals of epidemics to prevent their recurrence, and to secure for the population more healthy lives, whether it be in the towns or villages, or trade centres, or in the plantations, mines, and large works where the natives labour The two exceptions that have set themselves some approach to this standard, and are endeavouring gradually to reach it, are the West African Colonies and the Malay States.

Great Progress in West Africa.

We have an illustration in West Africa—though its sanitary progress has been set back temporarily by the War—that with even a small but well-trained Sanitary Service, supported by the Local Governments and encouraged by the Colonial Office, with the advice of the Medical and Sanitary Committee, there can be a great change effected in a comparatively short time. It is ten years since this Sanitary Service was created. In that time water supplies have been and were being introduced, drainage was being attended to, breeding-places of mosquitoes were destroyed, extensive marshes were filled up, building laws were enacted, town-planning was engaging the attention of the authorities, laboratories were established, and a special research laboratory was built in Southern Nigeria, and has been carrying out valuable investigations. A Commission on Yellow Fever has been the means of having the circumstances connected with outbreaks investigated, and the disease promptly dealt with. The result has been a healthier life for the European and a greater freedom from disease for the African. There is much more work to be done, the sanitary service has to be enlarged, and I hope that there will soon be a College of Medicine and Hygiene established, where the African can be educated in both these subjects, and be fitted to assist in the work of sanitation and in the medical treatment of his fellow countryman. Much of this success is due to the active and harmonious co-operation of the officials of the Colonial Office with the expert members of the Medical and Sanitary Committee, whose President has been for these ten years Sir Herbert Read, one of the Under-Secretaries of State for the Colonies, and the interest which the different Secre-

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taries of State have taken in the work. The function of the Medical and Sanitary Committee has been extended to East Africa. I should like to see it extended to the whole of the Crown Colonies, with suitable additions to its number of members, so that it might act as a Ministry of Health for them. The East and West African Colonies, which before the War were more than three-quarters the size of British India, and which by the additions since the War are larger than that Dependency, are not the only Crown Colonies that need supervision and advice in health matters.

Following on this, I should like to see a great Colonial Medical and Sanitary Service, the members of which, well trained in tropical diseases and their prevention in our Tropical Schools, should be available after good service and experience in one colony for transfer to others if required. Associated with this service should be a local service of native medical men and sanitary inspectors who have been trained locally, and whose knowledge of their compatriots would be of great assistance to the hospital physician, sanitary officer, and Government in their health administration and their campaign against disease.

I should like to see a league of Local Governments of the Crown Colonies having, through their representatives, conferences on health matters, from time to time, with the object of improving the conditions of life of the inhabitants under their charge.

An Imperial Health Museum in London as an Imperial Peace Memorial.

When His Majesty King Edward VII. died, I wrote to the Lord Mayor of London—who was chairman of a committee to consider a suitable memorial to commemorate his reign—I pointed out that His Majesty had always taken an active interest in the health of his people at home and in the Tropics. When Prince of Wales, he had been a member of the Royal Commission on the Housing of the Working-Classes. He had interested himself in the Commission to India to enquire into Leprosy, and he had been President of the International Congress of Hygiene, held in London in 1891. I ventured to suggest an Imperial Health Institute for Research with an Empire Health Museum, on a similar or larger scale than that of the British Museum or the Victoria and Albert Museum. My suggestion was not accepted at the time. Now that there will be an Empire War Memorial, worthy of the far-reaching victory which has secured safety for us all, I would suggest that, in addition to the War Memorial, there should be a Peace Memorial, in the form of an Empire Health Museum in London, worthy in every respect to illustrate the means by which we can triumph over preventable disease, which, in the long run, is far more destructive to human life and to health than even the greatest war.

The Empire Health Museum would contain models, plans and other arrangements for illustrating the causes of preventable diseases, the conditions under which they spread, and the methods by which they can be prevented; the diseases communicated by animals, insects and vermin; the diseases of occupations liable to occur in different industries, manufactures, trades, foundries, workshops, mines, plantations and large works, and the known methods of prevention. Side by side would be healthy houses, cottages, flats and huts, contrasted with those that are unhealthy both at home and in other parts of the Empire; slums and garden cities; town-planning and insanitary areas; schools healthy and unhealthy; foods and their adulterations; alcohol and tobacco and their effects in excess; infant and child welfare; mother craft; water supplies; clean and pure milk as contrasted with the dirty and adulterated article, etc. There would be tropical, industrial, domestic, personal, housing urban and rural, and preventable diseases sections. The museum would be open to all. The stonemason, the pottery and earthenware worker, the wool sorter, the workers in hides and horsehair and flax, the dairyman, the school-master, the housewife, the painter, the miner, the brass founder, the electrician, and the worker in any industry or trade, at home or abroad, should be able to go and see the risks, if any, to which he or she may be subjected, and the means by which they may be prevented. There should be sections to represent not only the British Isles, but also Canada, Australia, South Africa, India,

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Burmah, and our Crown Colonies and Protectorates. Lectures and demonstrations, illustrated by models and the cinematograph should be given, and there should be attached a Bureau of Information.

The museum would be a standing reminder to our administrators that next to the safety of the Empire is the health of the people.

Sir JAMES CANTLIE, having taken the chair during the reading of the paper, proposed a hearty vote of thanks to Professor SIMPSON for his very interesting address, and congratulated the Society on having such a celebrated hygienist as their new President.

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Obituary.

RIGHT HON. SIR WILLIAM MACGREGOR, G.C.M.G., C.B., D.Sc. (Hon., Cantab.), LL.D. (Edin.), LL.D., M.D. (Aberd.).

Not a few medical men have acquired distinction in other than purely professional spheres, but there are few who have done so and yet maintained their scientific acumen, as was the case with the subject of this notice.

Sir William was a typical instance of the "canny Scot," who has ever been in the front rank of pioneers of the Empire. He had the spirit of a Livingstone, caring little for the plaudits of men, but possessed of indomitable perseverance in any work to which he put his hand. After fifteen years spent in the Colonial Medical Service, he became Administrator of Papua, where he gained his reputation as an ideal administrator of a tropical colony.

It was in Lagos, however, that he shewed his determination to apply the teachings of medical science in the every-day life and work of a malarial region. It was fortunate that Sir William was appointed to Lagos at the time when the anti-malarial measures advocated by Major Ross (as he then was) were being strongly advocated, and the association of these two men proved to be an important factor in the promotion of sanitary reform in West Africa. He was so assiduous and thorough in carrying out every form of anti-malarial measures, that it is difficult to associate his name with any special theory of prophylactics. He drained swamps, fitted houses with wire gauze, strongly advocated the use of quinine as a prophylactic, and, above all, instituted a system of teaching on malaria to children in the schools. The children were provided with bottles, in which they were able to watch the development of larval mosquitoes, giving them practical demonstration of the life history of these insect pests and their relation to malaria, so that they became well acquainted with the health conditions of their surroundings. This system—which was made a fundamental part of the educational system in Lagos—was an object lesson which might well be followed in our own elementary schools at home in matters of personal hygiene.

The Ladies' League—which he instituted to preach the use of quinine and other sanitary measures—was a common-sense movement of great value.

Sir William was not content with studying this subject in West Africa, but he personally made extensive enquiries in Italy, and his despatch on malaria, as a result of that visit, may be regarded as one of the classics of early malarial literature following upon the discoveries of Laveran and Ross.

His breadth of mind and indomitable perseverance carried him through difficulties which would have checked many, and the Society of Tropical Medicine and Hygiene may count itself happy to have included his name in its roll of Honorary Members.

C. F. H.

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ANTI-MOSQUITO MEASURES IN PALESTINE DURING THE CAMPAIGNS OF 1917–1918.

BY

MAJOR E. E. AUSTEN, D.S.O.

Those of us who believe that in malaria, as in tactics, "the best defence is a vigorous attack," must have been greatly heartened by reading a few months ago in a medical paper that, for "the practical prevention of malaria . . . . the direct limitation of mosquitoes is the only proven measure of practical value. . . . ."* Without in any way presuming to take this statement for granted, or to trench upon matters which I am not competent to discuss, I hope it may not prove without interest if I endeavour to give some account of what was done during the Great War towards the suppression of malarial mosquitoes within our lines in Palestine, a country in which, as I need scarcely remark, all three forms of malaria occur, although malignant tertian is by far the most common.

For more than one reason (countless natural mosquito hiding-places in the shape of crannies and recesses in earth cliffs or among rocks; the enormous number of artificial hiding-places in Palestine, represented by subterranean cisterns, rock-tombs and deep well-shafts; and the practical impossibility under war conditions of dealing with the interior of native habitations) anything like systematic destruction of adult mosquitoes was out of the question. Consequently, apart from the issue in 1918 of mosquito nets

* "Anti-malaria Measures in England," by ANGUS MACDONALD, M.D., D.P.H., D.T.M., Temporary Captain R.A.M.C. (*British Medical Journal*, No. 3048, May 31, 1919, p. 669).

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and a repellent ointment of doubtful efficacy, efforts were concentrated exclusively on larval destruction and the prevention of breeding. Lest any who may happen to be specially interested should cherish unfounded hopes, let me confess at the outset that I have nothing that is novel to communicate. All that was done was on well-known lines, albeit, as I hope to shew, the measures taken were on a fairly extensive scale. Under active service conditions, the simplest measures alone are practicable, and in Palestine the vital importance of water, both to man and beast, precluded the use of larvicides, such as the Panama fluid or ialine, even had they been available.

On entering Palestine in March, 1917, the Egyptian Expeditionary Force as a whole had had no experience in anti-mosquito work. It is true that at Kantara, on the Suez Canal, in the autumn of the previous year, a large amount of highly valuable and successful work had been performed, in the shape of draining, training and reclaiming a considerable extent of the swampy, mosquito-infested south-eastern shore of Lake Menzaleh. These operations, however, under the able direction of Captain J. Inglis, O.B.E., R.A.M.C. (T.), and with the help of native (E.L.C.) labour, were carried out by the 92nd Sanitary Section, a unit which remained at Kantara throughout the campaign, and did not accompany the Desert Column in the forward movement across Northern Sinai, which commenced in September, 1916. But no sooner did the Desert Column reach the line of the Wadi Ghuzze, at the time the first Battle of Gaza (March 24th, 1917), than the need for anti-mosquito operations along many miles of front and on definitely organised lines became apparent, and the requisite experience forthwith began to be gained in the bitter school of necessity. Thereafter the story falls naturally into three periods, under which it may now be considered, corresponding to the three defensive lines successively taken up by our troops, namely:—

(1) Wadi Ghuzze Line, March 24th–October 31st, 1917.

(2) El Jelil—Wilhelma—Jerusalem Line, December 23rd, 1917–February 22nd, 1918.

(3) Arsuf—El Ghoraniyeh Line, March 22nd–Sept. 19th, 1918.

(1) Wadi Ghuzze Line.

During the whole time that our troops were held up before Gaza, that is from the end of March to the end of October, 1917, the Wadi Ghuzze was partly in our front line. In the southern section (from Tel el Jemmi to El Gamli) our line either followed the wadi or ran parallel to it at a distance of about one mile from the further bank. From Tel el Jemmi to the sea, that is in the section facing and outflanking Gaza itself, the actual front line was at a greater distance, namely from two to four miles beyond the wadi. But during the whole period the wadi formed the only source of water supply for men and animals, and fanatis (water tanks carried on camels) were filled and camels, mules and horses watered in it daily. Thus every day large numbers of men visited and were employed in the water-bearing reaches of the wadi, while parties engaged upon duties connected with water-supply bivouacked permanently on the wadi banks.

Throughout the period under consideration, water in the wadi was only present, or obtainable by digging, in certain portions, amounting in the aggregate to a total length of some eight miles. In each of these portions the original conditions, as found in the month of April, consisted in a series of shallow pools of clear water, varying in dimensions from a yard or two in area to 80 or 100 yards in length, by seven or eight yards or more in breadth, and either isolated and stagnant, or connected by shallow streams meandering through stones, shingle or sand. Both pools and connecting streams were choked with green alga *(Spirogyra)*, and all alike were literally teeming with Anopheles *(Anopheles turkhudi)* larvæ. In proof of the latter statement it is only necessary to mention that, at almost any point, it was possible to secure at least a dozen Anopheles larvæ, simply by thrusting a ladle at hazard into the alga mat. Under such conditions it was fortunate indeed that at that time the front line troops included no units already infected with malaria.

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For purposes of administration and mosquito-control, the eight miles alluded to were divided into four sections, each of which was dealt with by the Division occupying the corresponding section of the line for the time being. Work was carried out under the supervision of Divisional Sanitary Sections, the necessary working parties being supplied either regimentally, or, in the case of one Division (the 54th), by the Egyptian Labour Corps. The work done included oiling at five- or seven-day intervals (usually with a mixture of two-thirds heavy oil and one-third paraffin or kerosene, which was found to work well); weeding; training of pools; canalising connecting streams (in some cases the latter were converted into "Irish drains," which, however, are only satisfactory if completely covered with soil, so that the water is inaccessible to egg-laying mosquitoes, and there is no upward soakage); and the filling in of shallow extensions.

Owing doubtless to the high daily temperature in the wadi, alga after weeding had been carried out, and unless oil had been applied very thoroughly and conscientiously, was found to grow again with astonishing rapidity, three or four days being quite sufficient for the production of a fresh crop. In the climate of Palestine, green alga invariably connotes Anopheles-breeding, and its existence should always be regarded as a danger signal. Alga, however, which has been killed by oil is not usually a breeding-place. As regards oiling, the strong breeze, which blows daily up the wadi from the sea, from about 10 a.m until sundown, speedily drove the oil to the leeward end of pools, although not as a rule until it had served its purpose; for this reason it was found advisable to apply the oil either before the breeze had sprung up or after it had dropped. In the case of streams between pools, after canalisation, oil-bags (bundles of cotton-waste or tow, soaked in oil, wrapped in sacking and submerged with stones) were sometimes used, but were very liable to interference and consequently not particularly satisfactory.

In certain sections of the wadi, trouble was caused from time to time by the digging, either by troops or Egyptian camel-drivers, of small water-holes in the wadi bed. Such holes, which in damp spots may be scooped out by hand, are difficult to discover and easily overlooked, but are often a fertile source of Anopheles-breeding. Holes of this kind containing not more than 1 or 1½ gallon of water were found swarming with newly hatched *A. turkhudi* larvæ, and in one Division, by the posting of conspicuous notices in the vicinity of crossings over the wadi, a praiseworthy attempt was made to impress upon troops the danger of digging them.

A spot which for some time was especially troublesome was that known as Shellal, a very important crossing place over the wadi, and one of the chief centres of water-supply. The water, which was brackish, with an average salinity of some 200 parts of NaCl per 100,000 (204·6 parts in April, 198·76 parts in the middle of July), oozed freely from the further bank, and collected in small, alga-choked holes in the rock, which there crops out. *A. turkhudi* originally bred very freely in these holes, and eggs as well as larvæ in all stages were found in large numbers. The species mentioned deposits its eggs horizontally side by side in "ribands," the arrangement being very suggestive of a filled machine-gun belt. The ribands are soon broken up into short lengths by the wind, but once seen are quite unmistakable; a length of about one inch, examined on June 30th, was found to consist of ninety-four eggs.

In the month of June conditions at Shellal were largely altered by the construction, in the bed of the wadi, of a reservoir holding 900,000 gallons of water. This reservoir, formed by building a wall across the wadi bed, speedily filled and submerged a large number of what were previously bad breeding-places in the bed and further bank. Breeding, however, still continued for a time in holes and small bays in the rock at the upper end of the reservoir, but, by dint of filling in these cavities and repeated applications of oil, was eventually entirely suppressed. The growth of alga in the reservoir caused much trouble, but the labour of removal was facilitated by the use of a rough punt, constructed by R.E. In July a small consignment of larva-destroying fish (*Tilapia nilotica*, otherwise known as "Bulti"), from Egypt, was introduced into the reservoir. One hundred and five of these fish, about 1 or 1½ inch in length, somewhat resembling tiny perch, caught by Captain INGLIS in Lake Menzaleh, at Kantara, at 6 p.m. on July 11th, were despatched the same night by the military railway across the Desert

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of Sinai, and reached their destination on the following afternoon. Air had been pumped into the tanks at half-hourly intervals through the night, and, as a result of this precaution, the little fish stood their trying journey fairly well, and some eighty survivors were duly decanted into the reservoir, where they at once made themselves at home, and swam about in small shoals. Additional consignments were subsequently received, and some of the fish were introduced into a pool higher up the wadi, between Tel el Fara and El Gamli. The fish in the reservoir increased and multiplied, and throve exceedingly, but whether they fulfilled their intended purpose, or were in any way responsible for the disappearance of the mosquito larvæ, it was impossible to ascertain.

From May until October, each section of the wadi, in which anti-mosquito work was in progress, was subjected to a minute inspection at frequent intervals. The original condition as regards Anopheles breeding has already been described. As was almost inevitable, sets-back due to military exigencies, or fluctuations in the supply of oil, occurred from time to time, but on the whole, improvement was steady and continuous. Towards the close of the period under consideration, it became increasingly difficult to find half a dozen Anopheles larvæ, and in September and October, on more than one occasion, one or other section of the wadi was searched as thoroughly as possible without the discovery of a single larva. These facts may be left to speak for themselves.

The necessity for constant and unremitting care and labour in anti-mosquito work of this kind was, however, never more completely illustrated. The enemy was merely held at arm's length, and was quick to take advantage of the slightest lapse on the part of the defence. This was demonstrated repeatedly, when delays in getting to work occurred as the result of periodical changing over of Divisions; and a visit to the Shellal section shortly after the capture of Beersheba (October 31st, 1917), when the troops had gone forward and all anti-mosquito operations in the wadi had necessarily ceased, shewed that breeding had again begun where not a larva could be found some ten days before.

(2) El Jelil—Wilhelma—Jerusalem Line.

The occupation of this line coincided with the rainy season and the winter months (of 1917-18), a period not usually associated with malignant tertian fever. At the end of December, 1917, however, a sharp local outbreak of this disease—twelve cases, apparently primary, unfortunately resulting in five deaths—in the XXIst Corps, near Jaffa, concentrated attention on conditions existing in the extensive orange-growing area to the north east and east of the town in question. Although probably few people, unacquainted with the system in vogue, would naturally regard the culture of the Jaffa orange as being largely, if not mainly responsible for the local continuance of malaria, it speedily became evident that such is the case. For irrigation purposes, each of the orange groves, which extend for miles round Jaffa, is provided with a well, connected in most cases with a large cement storage-tank. The wells, which are very numerous, though presenting certain differences in detail, are similar in type, and of large dimensions. In each case the cylindrical shaft is from 12 to 15 feet in diameter, and the depth to the surface of the water, though sometimes not more than a dozen feet or so, is usually considerable, varying in different cases from 25 feet to 75 feet, or even more. The older wells, from which the water is raised by means of a "Persian wheel," are of the primitive kind seen in Southern Palestine, and are roofed over with an arch of stone. Wells of more recent construction are fitted with a bore tube, through which the water is raised by means of an oil pumping-engine, and many of the older wells have been converted to this type. Except in the vicinity of Mulebbis, where most of the well-shafts are exposed to the sky, the mouths of the more recent wells, fitted with mechanical pumps, open either into the engine room itself, or into a roofed shed of stone or mud adjoining. Thus in nearly all cases the well-shaft is protected by some kind of roof—a very important factor during the winter months from our present point of view, since not only is the nightly temperature in the shaft consequently higher than that of the outer air, but the surface of the water itself, as well as the inner wall of the shaft, is protected from the torrential downpours of rain which are of frequent occurrence during January and February. The result is that

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mosquito-breeding is able to proceed as usual during the season when it is quite impossible in the open, while the sheltered well-shafts afford safe retreats and hiding-places for the adult insects.

In January and February the entire area was subjected to careful inspection, in the course of which, so far as possible, every orange-grove well was examined, with the result that Anopheles-breeding, established by the presence of larvæ of all ages, from just hatched young to adults, was found in a large number of wells. In some instances pupæ also were discovered, while in many cases the adult insects, disturbed by the lowering of a bucket, issued from the mouth of the shaft in numbers. It not infrequently happened that adult Anopheles were dislodged in this way from wells in which no larvæ were found, but owing to the difficulty of making an exhaustive examination at the bottom of a deep and dark well-shaft, the absence of larvæ in these cases could not safely be inferred. The most striking instance of larval abundance met with occurred on January 18th, when the surface of a bucket of water brought up from a depth of 57 feet, from a dark well-shaft opening into an engine room in which the native caretaker, his wife and baby were living, was found to be literally alive with adult Anopheles larvæ. A rough computation on the spot shewed that the bucket contained from 150 to 200 larvæ at least. Shortly afterwards (1.0 p.m.) an adult Anopheles appeared in the engine room and bit a soldier on the neck. In the case of different series of wells examined during January and February, the presence of Anopheles, larvæ or adults, or both, was established in from about 20 per cent. to 40 per cent., the higher percentage being obtained on February 18th, in orange-grove wells in the vicinity of the Jaffa-Ramleh road. Anopheline larvæ were discovered in a small percentage of the storage-tanks connected with the orange-grove wells, although here, as was to be expected, since the tanks are fully exposed to the weather, breeding was far less in evidence. Moreover, many of the tanks were found to contain large numbers of small fish.

The species of Anopheles encountered was in all cases the same, *A. bifurcatus* (LINN.), a large, peculiarly aggressive and bloodthirsty mosquito, which also breeds freely in subterranean rock cisterns in Judea and Galilee, and is common in the British Isles and Europe generally. The behaviour of this species strikingly demonstrates the potential danger lurking in the popular belief that Anophelines bite only after sundown, for *A. bifurcatus*, when dislodged from its hiding-places, at once attacks and sucks blood greedily, even in brilliant sunshine, in the hottest hours of the day. This was seen over and over again in the course of the investigations described; men examining wells were bitten repeatedly, while female Anopheles were frequently caught with their abdomens full of partially digested blood. Personal experiment shewed that the bite of *A. bifurcatus* causes no pain or irritation whatever at the moment of infliction, and so may readily pass unnoticed. In this connection, an instance pointing to the danger to troops drawing water from unprotected orange-grove wells may be mentioned. At 2.50 p.m. on February 16th, at a village well in Yazur, near Jaffa, surrounded at the time by women and girls drawing water, two female *A. bifurcatus* issued from the well and were caught, one in the act of biting the leg of an Arab boy, who appeared entirely unconscious of the fact.

As soon as the breeding of *A. bifurcatus* in the orange-grove wells was definitely established, the whole area was divided into sections and charted, each well receiving a distinctive serial number, after which the wells were oiled periodically by the Sanitary Sections of the Divisions concerned. The same treatment was also applied to such storage tanks as did not contain fish. Wherever possible, sluices were opened and tanks allowed to run dry; certain orange-grove owners, however, raised objections to this practice, owing to the risk of tanks, which are large and expensive structures, often twelve or fifteen yards square, cracking in the sun when empty. In the case of the wells, since many were used for drinking purposes, either by troops or natives, it was deemed advisable to employ paraffin alone, unmixed with heavy oil. So far as could be judged from subsequent examination, the treatment adopted was entirely successful. A proposal to close the mouths of wells with mosquito-proof covers was mooted only to be forthwith abandoned, since, owing to the fact that these openings are partially blocked with

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irregular obstructions in the shape of pumping or other water-raising machinery, the provision of effective covers would have been practically impossible.

Until the closing week of February, 1918, the remainder of our line, with its right flank resting on or near Jerusalem, was still entirely in the Judæan hills, where at this season climatic conditions and the continuance of the rains rendered anti-mosquito operations impossible as well as unnecessary, so that we may at once proceed to—

(3) Arsuf—El Ghoraniyeh Line.

This line, which was held until the final general advance, which closed the campaign, was by far the most important of all, on account of its extent (some sixty miles from west to east, that is from the sea to the bridge-heads beyond the Jordan), the large number of troops holding it, and the highly malarious country through which it ran. The diversity of the physical conditions in the different sectors resulted in several different types of mosquito breeding-places, the number and extent of which were vastly greater than anything previously met with in the course of the operations. Consequently the effort and man-power required for the suppression of breeding were enormously increased. Starting from the west, the physical conditions alluded to included those in the coastal plain (held by the XXIst Corps), a region where water abounded in the shape of several extensive marshes; the second largest river in Palestine, which was fed by a network of springs concealed among rank vegetation; and, lastly, a number of narrow wadis extending for miles, all containing water, and in most cases originally so choked with jungle that the water was invisible. Next came the hills (held mainly by the XXth Corps, though the right flank of the XXIst Corps rested among them), a labyrinth of limestone rock, often rising to an altitude of upwards of 2,000 feet. The hills are intersected by a maze of narrow, rocky valleys, frequently containing boulder-strewn, weed-choked streams, in all cases breeding-places of the malaria-carrier known as *A. palestinensis*, while the subterranean cisterns in and about the countless hill villages afford similar facilities to *A. bifurcatus*. Lastly, on our right was the scorching Jordan Valley (occupied by the Desert Mounted Corps), some 1,200 feet below the level of the Mediterranean, and eight miles or more in width. The portion of the valley within our lines was crossed by several streams, issuing from the hills and making their way down to the river, and also included, besides other breeding-places, a particularly dangerous wadi, commencing in No Man's Land and intersecting our front line, and several extensive marshes or pools in the jungle fringing both banks of the Jordan.

After this short survey of topographical conditions, we may proceed to a brief summary of the chief work carried out in the different sectors, commencing with the coastal sector and the XXIst Corps.

Anti-Mosquito Work carried out in the XXIst Corps Area, River Auja region, during the Spring and early Summer of 1918.

Tel Abu Zeitun Marsh, near Mulebbis.—This was a water-logged tract of grass and reeds on the south side of the River Auja, measuring about 1½ mile in its longest extent, and varying from 200 to 500 yards in width. *A. sinensis* (Wied.), and *A. algeriensis* (Theob.), bred freely here in May, while later *A. palestinensis* (Theob.), was also met with.

Twenty acres of reeds were cut, and the entire marsh was drained into the River Auja by means of trenches dug by E.L.C. The main drain was 14,000 feet long, and varied in depth from 6ft. at the commencement to 1ft. 8in. at its junction with the river; there was a fall of 7ft. 6ins. Branch drains, 4ft. to 6in. deep, exclusive of "ducts," were 11,000 feet in aggregate length. The total amount of earth excavated, much of it in the form of semi-liquid mud, was 8,500 cubic yards. Up to June 30th, 1918, 50,000,000 gallons of water had been drained away, and work equivalent to 15,354 E.L.C. men-days had been done. Branch drains and ducts were oiled twice a week by the 3rd (Indian) Division.

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By August, Anopheles-breeding had been stamped out. Water was still running freely in the main drain, though except in this and branch drains and ducts, with the exception of a single small " pocket " holding about one quart, no exposed water was to be seen anywhere. Yet, although the surface of the marsh had thus become quite dry as the result of drainage and evaporation, at the end of the marsh near Mulebbis there was in places, amounting in the aggregate to some four acres, even in the month of August still much water beneath the superficial skin of grass and matted roots of herbage. In these areas the ground shook and undulated when walked upon.

Burak Leil.—Two marshes near the sea, some two and half miles north of the River Auja; dimensions of the northern marsh, 700 by 200 yards; dimensions of the southern marsh, 300 by 100 yards; average depth of water, 10 to 12 inches. It is interesting to note that the snail known as *Bullinus contortus*, the intermediate host of *Schistosomum hæmatobium*, was found in the northern marsh in considerable numbers early in May. Although no Anopheles larvæ could be discovered in either marsh at that time, breeding is reported to have begun before drainage was completed.

These marshes were dealt with by cutting the reeds growing in the water, and pumping the latter into the sea, through two pipes laid for the purpose. A large sump was dug, and the water drained into this by means of trenches, and then pumped out.

Drainage was commenced on May 14th, and completed on May 27th The estimated amount of water to be got rid of was 6,000,000 gallons; the quantity actually pumped into the sea was 4,800,000 gallons; thus evaporation, aided by the cutting of the reeds, disposed of 1,200,000 gallons.

Personnel, etc., employed :—R.E., 16; E.L.C., 330; pumps (motor and steam), 4.

Baharet Katurieh.—This was a very extensive marsh (open water covered with reeds), north of El Jelil, and about one mile from the sea. Its dimensions were 1,500 yards from north to south, by 500 yards from the west to east, and our wire ran across the centre.

The entire volume of water in the marsh was drained into the sea, through an ancient tunnel, some 150 yards long, under Argyle Ridge, and thence by a channel, approximately one mile in length, dug for the purpose. The tunnel, which had become blocked through falls of earth, was cleared, and the channel and an adit to the tunnel were dug by Sikh Pioneers and the 28th Punjabis. Two long drains were also cut through the marsh itself. The marsh was dry on July 10th.

At the end of April no Anopheles larvæ could be found in the marsh, but they are reported to have been abundant later on, when the water began to collect into pools. At the same time, imagines of *A. maculipennis* were said to have been present in large numbers in the bivouacs of the Sikh Pioneers, on the west side of Argyle Ridge, and also on the roof of the tunnel. On August 18th, however, when the Indian Infantry camp had been removed from the vicinity of the tunnel, no Anopheles could be found in the latter situation.

River Auja.—Although this deep and clear, rapidly flowing river was not in itself an Anopheles breeding-place to any considerable extent, a multitude of densely overgrown and concealed springs draining into the stream at and near its source at Ras el Ain was only too favourable to Anopheles development; while, as will be seen directly, even in the actual river breeding occurred in places where the necessary shelter was afforded by patches of floating weed or débris. The original condition of the river banks above the lower reaches, which were swept by sea-breeze, was such as to render it advisable to take no chances, and it was therefore decided to clear them.

The banks, which were thickly overgrown with high, dense vegetation, were cleared and trimmed from Ras el Ain down to Jerisheh Mill, a distance of some eight miles in a straight line, and much more along the windings of the stream. On the right bank of the river (No Man's Land), between El Mirr and Ras el Ain, where the ground was very swampy and full of springs, a very large number of small drains, leading into the river, was dug, and oiling parties were regularly employed At Ras el Ain itself it was impossible to employ more than half a dozen men at a time, since the spot was under direct enemy observation, and was shelled daily.

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The El Mirr—Ras el Ain region was originally a hot-bed of *A. palestinensis*, as well as, probably, of *A. sinensis*. In April and May no Anopheles nor other mosquito larvæ could be found in the river itself. On August 5th, *A. maculipennis* larvæ were discovered in some numbers in sheets of woody débris floating on the water at Hadrah Dam, one and a half mile above Jerisheh; also in closely growing patches of floating weed—almost exclusively a hard, brittle weed, dark brown on the surface and green beneath—present at certain spots near the banks between Hadrah and Jerisheh. Shortly before this, imagines of *A. maculipennis*, some of them gorged with blood, had been observed in tents on the right bank of the river, at and some 600 yards above Hadrah Dam. The banks and sluice-bays at Hadrah, which had already been dealt with some time previously, were again cleared and trimmed by half-battalion 125th Rifles; the sheets of woody débris referred to were removed by means of special scoops devised for the purpose, constructed of expanded metal and supplied by Ordnance. The patches of weed lower down were oiled and subsequently removed. Below Jerisheh, where the river is too open and wind-swept to be a likely breeding place in any event, none of the brown weed alluded to was seen.

Wadi Rabah, Wadi Ishkar, and one or two other similar Wadis.—Small wadis on the right bank of the Auja, near Ferrekiyeh, varying in length from about 200 yards to 1,000 yards or more. These wadis were originally choked with brambles, reeds and other high vegetation, completely concealing the water in them. The vegetation was entirely removed, and the watercourses were canalised, pools being filled in or reduced to narrow dimensions. In some places the banks, where weak and soggy, were revetted with eucalyptus branches. Such revetments, however, are unsatisfactory, since larvæ may shelter in their interstices, which are difficult to reach with oil. In the Wadi Ishkar, therefore, revetments of this kind were replaced by others formed of sandbags, which are much more suitable, and leave no interspaces. Whether sandbag revetments in water will endure for a whole season could not be determined.

In places oil (half heavy oil, half paraffin) drip-cans (small tins nailed to short stakes) were used, and worked well. It is worth noting that the hole in a small-sized tin should not deliver more than four or five drops per minute, otherwise the oil will soon be exhausted. The lower part of the Wadi Ishkar, where the channel was some six feet broad, was dammed, by means of sheets of corrugated iron, into compartments about 50 or 60 yards long, each with its drip-can.

Wadi Abu Lejja.—A much longer wadi on the left bank of the Auja, joining the river at Ferrekiyeh, and, like the small wadis previously mentioned, originally choked with brambles and other vegetation. The jungle was entirely cleared away, and about 6,000 yards of watercourse were filled in. This was done by throwing down earth from the banks, so as partially to fill up the bed, and then digging a trench down the middle, the trench subsequently being filled in. During the last week in April, 1,100 E.L.C. were employed in this wadi.

Wadi Barideh.—Commencing between Yazur and Selmeh, this wadi, which is larger than any of the foregoing, runs past Sarona and eventually joins the Auja on its left bank about half a mile below Jerisheh. In the upper part of the wadi, above the Jaffa-Mulebbis road, 2½ miles of pools were filled in, and the remainder of the watercourse, after being reduced to narrow dimensions, was canalised. Below the Jaffa-Mulebbis road, the margins of the watercourse were trained for about half a mile. From the point at which training was stopped to the Auja the stream is much broader, and the banks in this portion were trimmed and cleared of vegetation in May. By the middle of August the vegetation had grown up again to such an extent that there was little sign that anything had ever been done, and a gang of some 300 E.L.C., previously working on the part of the wadi above the Jaffa-Mulebbis road, was employed to carry out clearing and weeding afresh.

Wadis in the Hills.—In the hills, streams in some half-dozen small wadis were canalised.

Species of Anopheles met with.—In the XXIst Corps area no fewer than seven species of Anopheles were met with, namely, *A. bifurcatus* (LINN.), *A. algeriensis* (THEOB.),

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A. maculipennis (MG.), *A. sinensis* (WIED.), *A. pharoensis* (THEOB.), *A. mauritianus* (GRANDPRÉ), and *A. palestinensis* (THEOB.) With the exception of *A. pharoensis*, all these were found breeding.

ANTI-MOSQUITO WORK CARRIED OUT IN THE XXTH CORPS AREA.

The XXth Corps, being entirely in the hills, was in the fortunate position of having no marshes and no large river basin to contend with. Consequently no engineering tasks on an heroic scale, such as those just described, were called for, and the amount of anti-mosquito work to be done in this, the central, sector of our line was far less than on either flank. Nevertheless there were many small mountain streams to be dealt with, weeded and canalised so far as their rocky beds would allow, while, as already indicated, the underground cisterns in the hill villages presented a special problem of peculiar difficulty. Instructions for the conduct of anti-mosquito work were issued by Corps Headquarters well in advance of the breeding season in the open. The system adopted was that of anti-mosquito squads—one N.C.O. and six men per battalion, proportionately fewer men in batteries and smaller units for investigation and the rendering of weekly reports, while work on the streams was carried out by regimental working parties. An order prohibiting bivouacking within 600 yards of any village was issued, but in practice proved difficult to enforce. A weekly inspection of anti-mosquito work in each Divisional area was carried out by Capt. (afterwards Major) S. F. ST. J. STEADMAN, D.A.D.M.S., XXth Corps.

Wadis.—The streams in a number of wadis, such as the Wadi Hamis and the Wadi el Jib, were cleared and kept free from weed, and were canalised wherever canalisation was possible. Work was carried out chiefly by working-parties of Indian Infantry, and results as regards the suppression of breeding of *A. palestinensis* were satisfactory. In one case, near the village of Ain Arik, ten miles north-west of Jerusalem, shallow puddles in hollows in flat rocks were found teeming with egg-ribands and larvæ of the species mentioned, and were promptly got rid of by sweeping. It need scarcely be remarked that in many places, owing to the rocky nature of the channel, anything like actual canalisation was extremely difficult if not impossible. In other cases, where the bed of the stream was softer, difficulty was sometimes experienced in inducing Indian troops not to use large stones for edging purposes. Such margins look neat, are conspicuous evidence of work done, and seem naturally to commend themselves to the Indian soldier. Owing to the inevitable interstices they are, however, a dangerous delusion, and should never be employed; low ramparts of smooth earth are what is required.

Village cisterns.—In the majority of the hill-villages in Judæa (as was also the case in Jerusalem itself until June, 1918, when a permanent water-supply was introduced into the Holy City by the British Army), the inhabitants are dependent for their water upon subterranean cisterns of various types hewn in the rock, and filled during the rains either from the roofs of houses, or by drainage from the land. In Jerusalem, according to Professor MÜHLENS (1913), there are at least 5,000 to 6,000 of these cisterns, and perhaps even more, and most if not all of the villages within the XXth Corps area contained a proportionate number. These cisterns, which in at least 50 per cent. of the cases, and probably more, are breeding-places of *A. bifurcatus*, form the real crux of the problem of mosquito suppression in Judæa. It having been asserted that the inhabitants would object to the application of oil to their water, on March 12th, 1918, an interesting experiment was made in order to test the truth of this statement. For this purpose Râm Allah, a large Christian village some nine miles north of Jerusalem, was selected; the inhabitants had previously been apprised by the mayor of the intended operation and its object, and no opposition was shewn. In Râm Allah there are said to be more than 100 cisterns, fed chiefly by rainwater from the roofs of houses. These cisterns have narrow shafts, and mouths (often only wide enough to admit a bucket and frequently protected by hinged covers of iron) opening sometimes inside houses, though more frequently in the compounds adjacent thereto. The diameter at the water level varies greatly, from 3 or 4 feet to as much as 7 metres, or more. Since the interior of the cisterns is in all cases dark, and only a small portion of the water surface can usually be seen, it is very difficult to

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estimate the amount of oil required in each case. At first the oil was applied by means of a sprayer; this was found to be unnecessary, and the paraffin was simply sprinkled from a cigarette tin; the oil atomised well, and produced a satisfactory film. A bucket lowered into the first cistern visited, the opening of which was inside a living-room in a native house, brought up ten young and adult larvæ of *A. bifurcatus*; a pupa of the same species was found in another cistern, with mouth in the open air, but as a rule, owing to lack of time, the cisterns treated were not examined for larvæ. Imagines of *A. bifurcatus* issued from very many of the cisterns visited; as a rule females alone were seen, but one cistern (mouth in open air) contained four or five males, as well as a number of females. In all, 29 cisterns were oiled; three of these were not examined; larvæ or adults of *A. bifurcatus* were present in 14; 12 (including some not examined for larvæ) shewed no adults. It is perhaps worth mentioning that an inhabitant of Râm Allah, a Syrian medical student, on being shewn living larvæ of *A. bifurcatus*, said that they had nothing to do with malarial mosquitos, and were to be found in every cistern in the village!

Four days later it was reported by Capt. K. B. WILLIAMSON, R.A.M.C., O.C. 53rd Divisional Sanitary Section, that the results of the experiment were "satisfactory." "All the people complain," wrote Capt. WILLIAMSON, "of a bad taste in the water, and many have drunk from other cisterns, or water stored at the time of our visit; but yesterday they were drinking from the oiled cisterns, and reported the taste 'much better.' They all took the oiling in good part, and several returned thanks for it! It must not be forgotten, however, that the people in this village are all Christians, and that the standard of education is exceptionally high. . . . The oil appears to have effectually disposed of large numbers of the larvæ, and I failed to find adult mosquitoes in any of the cisterns where we had noticed them, though the covers had been kept on. From ocular evidence, some at least were dead on the surface of the water; and although it is too early to be sure of this, it seems as if the adults were practically all killed. Only continuous observation can establish this, as I have often noticed that cisterns which contain Anophelines on one day in the shaft yield none on another, and in bad weather, such as we have been having, they keep below. The contrast between yesterday and the day of the experiment was very marked however, and there is little reason to doubt that the oil has been effective in destroying adults." Capt. WILLIAMSON added that, from observations made by one of his N.C.O.'s, he considered that, at the period mentioned, from 60 per cent. to 65 per cent. of the cisterns in Râm Allah were infested with mosquitoes.

In order to test the attitude of the inhabitants of a non-Christian village towards oiling, on March 27th a visit was paid to Shafât, a small Arab village about two miles north of Jerusalem, and a few hundred yards west of the Nâblus road. The mukdar, to whom the object of the oiling was explained, made no opposition whatever, and seemed quite pleased that oil should be applied to the cisterns. Four cisterns were oiled in the village, and seven others outside. *A. bifurcatus* larvæ (young and adult) were found in three out of the four cisterns in the village, and in two out of the seven in the open country. In one cistern in a compound in the village, *A. bifurcatus* and *Culex pipiens* (larvæ and adults in each case) were present in very large numbers.

On the following day at Selwâd, an inhabited village on the top of a steep hill, fourteen miles north-north-east of Jerusalem, and two miles in rear of our front line, ten cisterns were examined, and adults of *A. bifurcatus* were found in eight (very numerous in four). In one or two cases, between 1.0 p.m. and 2.0 p.m., the insects on being disturbed and issuing from the cisterns tried to bite. *A. bifurcatus* larvæ (two young specimens) were found in only one cistern.

Some three-quarters of a mile from Deir Ibzia, fourteen miles north-west of Jerusalem, was a group of cisterns, used by the villagers as a reserve supply at the end of the dry season, in all of which *A. bifurcatus* was very numerous and breeding. By way of experiment, the openings into these cisterns were closed with sacking plastered down with mud, but results were unsatisfactory, since cracks developed in the mud, and the covers were displaced by natives.

As the result of the experiments at Râm Allah and Shafât, orders were issued throughout the XXth Corps area for the periodical oiling of all wells and cisterns in close

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proximity to troops, and this was regularly carried out On the Mount of Olives, within half a mile of Corps Headquarters, some eighty cisterns were located and numbered, and the odd and even numbers were then oiled each week alternately.

In the official *Report on Malaria in the Egyptian Expeditionary Force during 1918*, it is stated that "There can be no doubt that the careful and systematic oiling of the wells and cisterns in all native villages must have saved an enormous number of cases, as there was an abundant reservoir of the malaria parasite in the inhabitants."

Anti-Mosquito Work carried out in the Desert Mounted Corps Area (Jordan Valley).

The anti-mosquito work accomplished in this notoriously unhealthy area was second only in extent to that performed by the XXIst Corps, while the climatic conditions under which it was done were infinitely more trying. Three species of Anopheles—*A. turkhudi*, *A. palestinensis*, and *A. maculipennis*—had to be contended with Of these, *A. turkhudi* was found breeding in enormous numbers in the Wadi el Mellahah, and in places almost certainly made its way into our lines from No Man's Land. *A. palestinensis* bred freely in the Wadi el Kelt and along the margins of several other streams, as also in irrigated land at Ain es Sultan, near Jericho, and in a swamp on the farther bank of the Jordan at El Ghoraniyeh. The chief breeding-places of *A. maculipennis* were pools in close proximity to the River Jordan.

Wadi el Mellahah—Rising in a swamp near the Jordan, some two miles beyond our front line, this wadi followed a southerly course, and, passing through our wire, joined the Wadi el Aujah near the junction of the latter with the river. A visit of inspection on May 26th shewed that, in order to render this part of the line tenable without a serious rate of invaliding from malaria, a very large working party would be necessary A small swampy valley just beyond our wire, filled with high reeds and oozing with stagnant, brackish water (apparently heavily charged with magnesium chloride), was literally teeming with larvæ and eggs of *A. turkhudi*. Conditions below and above this spot were almost equally bad, while the stream flowing down the wadi was choked with weed in places, and in urgent need of canalising and training throughout. It was considered that, to carry out requisite operations with the necessary promptness, a working-party of 1,000 men was needed, and a memorandum to this effect was forwarded to General Headquarters on May 27th. Three days later 1,000 men were at work. Canalisation and drainage were commenced on May 30th by Australian Light Horse and Indian Cavalry, under the able and energetic supervision of Capt. Brisbane, Australian R.E.; work was continued by Yeomanry (5th Mounted Brigade), in relief of A.L.H., and later by Indian Cavalry alone. On May 31st remarkable progress had already been made; the stream flowing down the part of the wadi within our wire was in course of canalisation; a marsh just short of the wire had had a drain cut through it, and was rapidly drying up; while in the small, sloping valley already described, the high reeds had been burnt off, and a drain had been cut down to the stream in the main wadi. Later investigation shewed that Capt. Brisbane's confident anticipation that the small valley would speedily dry up by evaporation was completely justified. So thoroughly was the work done, that when the Commander-in-Chief paid a visit of inspection to this part of the wadi, on June 4th, no Anopheles larvæ could be found. Canalisation was continued, wherever necessary, through very difficult, scrub-covered country for more than two miles beyond our wire; part of this distance, however, was occupied by a large open pool, which was full of small fish, and in which no breeding was possible. Beyond the confluence of the Wadi er Risheh the area was completely exposed to enemy observation, and finally, as was inevitable, the working-party of Indian Cavalry here employed came under shell fire, suffered a number of casualties, and had to be withdrawn. By this time, however, all that was possible had already been done, and maintenance was all that was required.

Wadi el Kelt, Wadi Nueiameh, Wadi el Aujah.—The streams in all of these were prolific breeding places of *A. palestinensis*, though the Wadi el Kelt (said to be the Brook Cherith of the Bible) was by far the worst. So dangerous were conditions here, that an

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Australian Receiving Station, which had been pitched near the deep, rocky gorge through which the stream flows before it leaves the hills, and close to the point at which the Old Jericho Road enters the plain, was moved to a safer though waterless spot some two miles lower down the Valley.

Owing to the rocky nature of the bed of the Kelt in the gorge, anything like complete canalisation of this part of the stream was impracticable; canalisation was done wherever possible, however, and the work was pushed for a considerable distance into the hills. In the Valley itself the bed of the watercourse is shallower, and in summer the stream either disappears altogether in places, or is represented by isolated pools. Both stream and pools were found to be teeming with small fish (several species), which were prevented from attacking the Anopheles larvæ by felted masses of weed. Here canalisation as well as weeding was possible, and both were promptly carried out with entirely satisfactory results.

The necessary operations in the other Wadis mentioned, though very extensive and laborious, presented less difficulty, since the streams were more open, and the training of their margins was easier; the major part of the task of working-parties consisted in filling in or otherwise obliterating, shallow, semi-stagnant, weed-grown expansions, which were the chief breeding-places. The upper part of the Wadi Nuciameh was blocked by high reed and bamboo jungle, which was burnt off.

Ain es Sultan.—An area several acres in extent, about 1½ mile north-west of Jericho, consisting of banana plantations and other cultivated ground, copiously irrigated by the overflow from two reservoirs filled by a perennial spring. This spot, which supplies Jericho itself with water, and was freely drawn upon by troops throughout our occupation, was a breeding-ground both of *A. palestinensis* and *A. maculipennis*. Breeding was suppressed, and swampy ground obliterated by stopping the irrigation of the bananas, repairing the broken banks of water-courses, and cutting shallow, straight drains wherever required. The necessary work was carried out by working-parties supplied by Yeomanry, but here, as everywhere else, once the original tasks were completed, constant attention and maintenance were required.

Owing to the small number of troops in the Jericho area itself, and the consequent difficulty in obtaining working-parties of the requisite strength, a contingent of E.L.C. was called for and supplied; 1,000 of these men being employed for a fortnight, and subsequently 600 for a period of two months.

Swamps and pools on the banks of the Jordan.—There were two large swamps on the east bank of the river, one close to El Ghoraniyeh bridge, the other some six miles to the south, at El Henu ford. Both were very bad breeding places, and were drained. At El Ghoraniyeh the work was carried out by Indian Infantry, under the supervision of Major WHITE, I.M.S. To make the work more completely effective, the Nimrin stream, which was the cause of the swamp, was diverted from its course; oiling also was subsequently employed. Through the large swamp at El Henu deep ditches were dug.

On the west bank of the Jordan, in the scrub and reed jungle, between El Ghoraniyeh and Makhadet Hajla, were several large pools of muddy water, dotted about on the surface of which were circular patches (two to three yards in diameter) of floating weed, in which, at the end of May and beginning of June, *A. maculipennis* larvæ were abundant. At the same time many imagines of the same species, some gorged with blood, were caught in the lonely monastery known as Kasr el Yehûd, in which a detachment of Indian Cavalry was quartered, and there could be little doubt that the insects came from the pools in question. The larvæ were confined to the patches of weed, which were of two kinds—one (the commoner) grassy, the other coarser and brittle, and probably of the same species as that already mentioned in speaking of the River Auja. When seen, the pools were evidently shrinking, and shortly afterwards they were reported as having dried up completely.

LINES OF COMMUNICATION.

Time precludes anything more than a passing reference to the large amount of anti-mosquito work carried out during the summer of 1918 on the Lines of Communication,

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but mention may be made of Latron, where, in close proximity to the Jaffa-Jerusalem road, several miles of a small stream ifnested by *A. palestinensis* were cleared and canalised by E.L.C. personnel.

LECTURES.

Whenever and wherever it was possible to arrange for gatherings of medical officers, lectures on mosquitoes, their life-history and the best methods of mosquito-control under active service conditions, were given. On these occasions, the general lesson that it was endeavoured to enforce was that it was the bounden duty of every unit to leave its area in a better condition than that in which it was found.

While apologising for the many shortcomings and omissions in the foregoing all-too imperfect sketch of what was done to protect our troops in Palestine from malaria, by the suppression of mosquito-breeding, with which this paper is alone concerned, the author desires to refer those who wish for fuller information to the official "*Report on Malaria in the Egyptian Expeditionary Force during 1918.*" It is hoped, however, that enough has been said to shew that an honest and whole-hearted attempt was made to cope with the problem presented. "Nature" is proverbially difficult to "expel with a pitchfork," and nowhere is this more true than in Palestine. But all concerned, from D.D's.M.S. downwards, worked with a will, while for the zeal and energy of the officers and other ranks actually engaged in the operations described no praise could be too high. Definite conclusions as to the results achieved in the actual prevention of malaria must necessarily be somewhat difficult to draw. Suffice it to say that, for the six months prior to October, 1918, the percentage incidence of malaria for the three Corps of which our front line was composed was just over five. To this, the sudden rise in the malarial rate within three weeks after the date of the general advance affords a sharp and significant contrast.

APPENDIX.

A FEW RECOMMENDATIONS WITH REGARD TO ANTI-MOSQUITO WORK ON ACTIVE SERVICE.

1. *Sanitary Sections.*—Sanitary Sections, attached to Divisions in the field, will always be called upon to supply the matériel required for anti-mosquito work. They should, therefore, be self-contained, and, in a malarious country, should take the field fully equipped with a reasonable amount of such stores as are likely to be demanded of them, which they should be able to replenish from an advanced base sanitary dump, in charge of a sanitary officer. Highly dangerous delays in getting to work on mosquito breeding-places are very liable to occur when, as at present, Sanitary Sections are obliged to indent on R.A.S.C. for oil, on Ordnance for rakes or scoops, etc.

As regards peace-time training, the practicability might be considered of affording to Sanitary Sections, liable to have to take the field in malarious countries, some practical instruction in the best methods of mosquito control. For training purposes, if necessary, use might be made of carefully prepared *models*, but the course should include practice in finding and recognising Anopheles larvæ.

2. *Malaria Officers.*—Whenever troops are in permanent occupation of a line running through malarious country, a *Malaria Officer* responsible for the execution and maintenance of anti-mosquito work should be appointed for each Divisional or Brigade area. The same officers should remain on duty throughout the period of occupation, and should not be changed or withdrawn when their Divisions or Brigades are relieved.

3. *Inspections.*—In anti-mosquito work, such as that described in the foregoing pages, provision should always be made for frequent inspections by a competent officer

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not actually engaged in carrying out the work. Officers employed upon the work may sometimes overlook details owing to being too close to them.

4. *Co-operation between R.E. and Medical Services.*—The next edition of *Field Service Regulations* should contain an injunction to the effect that, whenever troops are in permanent occupation of a line running through malarious country, due notice of any intended work affecting exposed water must be given to the D.M.S. or his deputy by the C.R.E. or C.E. concerned.

Discussion.

Dr. Andrew Balfour: I would like to pay a warm tribute to the excellence of the work carried out by Major Austen, and his colleagues, on the Palestine Front. In the summer of 1918, General Allenby, then in Egypt, kindly asked me if I would care to see the anti-mosquito operations being conducted in Palestine. Naturally, I said I would be delighted. and, in July of that year, I was able to spend a few very interesting days on the Palestine Front. Thanks chiefly to the facilities afforded me by Major Manson-Bahr and Captain Bengerfield, I was able to range from the Jordan Valley to the Mediterranean, and so was able to get a good idea of what was being done in the different areas.

As regards the Jordan Valley, Major Austen has spoken of the trying conditions under which the work had to be carried out, and I can assure you he has not exaggerated them. I spent only a single day in the Jordan Valley, but it was one of the toughest days I have ever had in my life. The heat was intense and the dust was terrible. As we were speedily soaked through with perspiration, the dust clung to us like a mantle, and I remember when we returned to Jerusalem, Major Austen resembled nothing so much as Lot's wife when she was turned into a pillar of salt. I saw much of interest in the Jordan Valley.

Close to the Dead Sea we examined certain pools of clear, slightly brackish water, in which no larvae could be found, presumably because they were full of small and very active fish. I would like to ask Major Austen if it has ever been determined to what genus and species those fish belonged. They were probably Cyprinodonts, but at that time had not been definitely determined.

You have seen a picture of the Upper Wady Kelt. This, I believe, is the place where Elijah fed the ravens in the olden days. Doubtless, unwittingly, he fed the mosquitoes as well, but if he had been there in July, 1918, he could not have done so, because the work of drainage and canalisation, which Major Austen has described, had effectively banished all mosquitoes so far as could be determined. There is little cultivation in the Jordan Valley, but there are small irrigated areas in the neighbourhood of Jericho. In one of them was a banana grove, the trees growing amongst rich mould. Here in an exceedingly tiny collection of water, so small that one would scarcely expect to find any larvae in it, we discovered those of *Anopheles palestinensis*. It was clear that no one but an expert like Major Austen would have been likely to examine such a minute nursery for anopheline larvae, and hereby hangs a tale. Major Austen on a previous visit had determined the anopheline breeding-places in this area and had demonstrated them to the men of the unit which held this locality, so that they knew them well and were able to take the necessary precautions. Major Austen, however, had unfortunately to depend for getting about on any chance means of transport which came in his way. As a result he had been unable to pay a second visit to this area. The old unit left and a new unit took its place. This new unit, not having been instructed in the peculiarities of anopheline breeding-places, did not discover these obscure nurseries, and so the mosquitoes had again got a footing. It is very necessary that anyone engaged on anti-mosquito operations in a country like the Palestine front should be able to get about rapidly and without delay. I am glad to say that a representation made to General Allenby resulted in Major Austen being immediately supplied with a motor-car which, however, he should undoubtedly have had all the time.

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The only other point to which I would refer is what I saw near Mulebbis on the Auja river Those engaged on anti-malarial operations were not only exposed to discomfort and to the dangers of malaria, but were also subjected to the perils of war. Shortly before I visited the Auja region, several of the workers near the springs of that river had been killed and wounded by shell fire. When I was there, Colonel BROWSE, I.M.S, who had sanitary charge of this district, took me across the river into No Man's Land We ran practically no danger, for we were screened from the view of the Turkish batteries by dense orange groves, but I mention the matter because, in many places, it was necessary to wage war against the mosquito in country beyond the British front lines. Hence, those engaged in the work were liable to be killed or wounded. In the orange groves, of which I have spoken, we found much the same conditions as in the banana grove near Jericho, for here again anopheline larvae were present in very tiny water collections unlikely to be searched except by an expert and hence a suspicious observer.

In conclusion, I would like to say that I think in the future the anti-mosquito work carried out on the Palestine front, the success of which was largely due to the sermons and exhortations of Major AUSTEN, who did all he could to make everyone keen and interested in the operations, will be cited as a classical example of what can be done under difficult conditions and in the face of the foe, and great credit is due to all concerned. There can be no doubt that their labours very materially assisted in bringing the Palestine campaign to its glorious and satisfactory termination.

Surgeon-Commander L. M. MORRIS, O.B.E., R.N.: I would like to give an account of two years' anti-mosquito work at the various aerodromes and airship stations in the Ægean Islands, where malaria had been prevalent in 1916.

The work consisted, firstly, in placing the camps in the most favourable defensive positions against mosquitoes, and then carrying out an active anti-larval prophylaxis in the vicinity.

The work was done by Greek labour parties, recruited from the neighbouring villages, and consisted in the drainage of swamps and marshland, after previously clearing the area by burning operations.

Malaria was severe along the north coast of Thasos in 1916. The local drainage of the marshes at Thasos, Kassandra, and at Mitylene produced most satisfactory results, and also reclaimed large areas for cultivation. At Imbros the work consisted in the canalisation of the river bed and streams.

Mosquito-proof huts were erected for all ratings, and careful personal precautions were taken when dealing with sentries and others exposed after sunset.

The results of two years' continuous work proved the great value of active anti-larval measures in maintaining the health of the personnel, and I consider that work of this nature, carried out on the well-known principles of clearing, drainage, and oiling, is the only method likely to give success when engaged on active service in malarious localities. The camps being stationary ones, it was possible to extend the anti-mosquito work at each successive year, and so to ensure good results as long as the work could be accomplished by native labour and did not require expensive outlay

For instance, at Kassandra, it was possible to completely eliminate mosquito breeding, within an area of two miles from the camp, by the complete drainage of the marsh, and by locating all the hidden springs, which were drained to the sea.

Malaria exists in the Ægean Islands as the result of neglect to provide proper outlet for the winter flood-water, which settles in extensive marshes near the beach and densely overgrown with reeds and bushes. The native population are freely infected, and constitute reservoirs of infection in camps if placed near the villages.

Dr. MANSON-BAHR: I should like to congratulate Major AUSTEN on his paper, and more especially as I know we can count upon the fact that the work he has done was even more effective than he would have us believe. As I had the honour to be more or less associated with him in Palestine, I should like to say that we regarded him as our

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guide, philosopher, and always our friend. His tall form and long stride were familiar to most in the E.E.F. from Jerusalem to Jericho, from Haifa to Helouan, and from Sela to Beirut, but I understand he was not popular with those officers who had an aversion to foot-slogging or being only secondary, perhaps, in an anti malaria organisation.

In larvicidal measures it is necessary to have efficient means of microscopical diagnosis at hand, sufficiently mobile to be of practical value under active service conditions. Such units were formed in the E.E.F., and were known as Malaria Diagnosis Stations, whose function it was, in addition to diagnosing microscopically all suspected cases of malaria, to indicate at the same time the localities whence the infection was being carried, so as to enable Major AUSTEN and his myrmidons to take the necessary action. I think it must be admitted that such an organisation is a necessary corollary of the other.

Major AUSTEN has not told you quite the whole story—someday, I hope, that will be written. How effective the anti-malaria measures in the stationary line were, was seen directly an advance took place on the night of the 19/20 Sept., 1918, when we went forward into country completely unsanitated in the malarial sense. After an incubation period of 9 or 10 days, large numbers of the most serious forms of malignant malaria began to run down the line. The figure rose to 1,800 new malignant infections, diagnosed microscopically, and the deaths from this cause from three or four a week to 300 or 400 in October, 1918.

This, then, was Major AUSTEN's contribution to victory: that by his example and precepts, and the actual work he performed, he assisted in keeping Allenby's force healthy and active through a malaria season in one of the most highly malarious countries in the world, and we now know what would have happened to this force had not the matter been taken in hand in time.

Major ALDO CASTELLANI: Major AUSTEN, in the very interesting paper he has read, clearly shews the paramount importance of anti-mosquito measures—using this term to cover, of course, also anti-larval measures—in the prevention of malaria in troops fighting in subtropical and tropical countries. I think we all agree that such measures, based on the great discoveries of MANSON and ROSS, form by far the most efficient method of prophylaxis. In my experience, however, in practice, it is always of great advantage to carry out at the same time a certain amount of quinine prophylaxis. In my inspections of military camps in malarious districts during the war, I have always noted that the least number of malarial infections occurred in those camps in which the two methods of prophylaxis were associated. But if quinine prophylaxis is useful in stationary camps, it is, in my opinion, essential in troops taking part in a war of movement, and we had ample proof of this during the rapid advance made a little over a year ago by the Italian Army, in conjunction with the brave British and French troops, from the Piave to the Isonzo and further north. The brigade of Marines, in which a most strict quinine prophylaxis was carried out during the whole period, thanks to Surgeon-General F. RHO, suffered from malaria infinitely less than the brigades of Army Infantry fighting under exactly the same conditions, but having practically abandoned quinine prophylaxis.

Of course, as is the case with everything else, if we want quinine prophylaxis to give good results it is necessary to carry it out in a thorough and scientific manner. It is not much use giving the men a few quinine tablets, and telling them to take one or two a day. They will take the first opportunity to throw them away.

Quinine should be given in adequate doses, and the medical officer should take care that it is actually taken. In badly malarious zones, such as the Balkans, five grains a day are not sufficient; at least ten grains a day should be given. To see whether the quinine is taken or not, Tanret's reagent will be found extremely useful. If the urine, after adding a few drops of this liquid, remains clear, one can be practically certain that quinine has not been taken.

In conclusion, I should be sorry if I have given the impression that I consider quinine prophylaxis to be more important than, or as important as, anti-mosquito prophylaxis; on the contrary, I believe the latter to be by far the more important of the two; but I also firmly believe that, in practice, the best results are obtained when the two methods are combined.

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TRANSACTIONS

OF THE

SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

JANUARY 16TH, 1920.

VOLUME XIII. No. 5.

Proceedings of a Meeting of the Society held on Friday, January 16th, 1920, at 8.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Professor W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P. (*President*), in the Chair.

REVISION OF THE LAWS.

The PRESIDENT submitted the following resolution :—

> That the Draft of the Laws of the Society as revised by the Council on 22nd of October, 1919, be approved, and that the new Laws come into force on the 1st of April, 1920.

Dr. C. F. HARFORD proposed, and Surgeon-Captain P. W. BASSETT-SMITH seconded the adoption of the resolution.

There was no amendment, and on the resolution being put to the meeting it was carried *nemine contradicente.*

Dr. P. MANSON-BAHR then opened a discussion on "Bacillary Dysentery."

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BACILLARY DYSENTERY.

BY

DR. PHILIP MANSON-BAHR, D.S.O., M.D., M.R.C.P.

The subject of bacillary dysentery has now become one of such magnitude, with ramifications extending into the sciences of bacteriology and pathology, that it cannot be dealt with in the limits of one paper, and what is most required at the present moment is a review of the subject as it stands after five years of strenuous warfare. One of the chief lessons borne home from the war in the East, has been the necessity of maintaining a staff of dysentery experts, both from the point of view of the diagnosis, as well as treatment of the disease. It is hardly necessary to point out that the primary diagnosis of dysentery must always remain in the hands of the bacteriologist, and that this person should be competent in the first instance to differentiate between the two main forms.

I need hardly remind this Society of the enormous wastage which has been caused, and the uncertainties which existed in the early stages of the war, owing to misconceptions in the differential diagnosis of bacillary from amœbic dysentery, and the almost lamentable lack of knowledge of the bacteriology, pathology and treatment of the former. I think this Society would be conferring a public benefit, were it to form the medium by which many such misconceptions could be cleared away.

EPIDEMIOLOGY.

It has been my experience throughout the war in Egypt and Palestine that the bacillary variety has been by far the predominating form included under the head of dysentery. The private records which I have kept shew that over 90 per cent. of cases examined during life and after death were bacillary in origin. It occurred most usually in epidemic form as a disease of the late summer and early autumn, though minor outbreaks may occur during the spring months. The observations which I originally made in Fiji, in 1910, on the seasonal incidence of bacillary dysentery and the relationship which seemed to exist between it and the house-fly (*Musca domestica*), have been amply borne out by subsequent observations in Gallipoli, Salonika, Egypt and Palestine, where more intimate knowledge of the life-history of that insect has thrown a flood of light upon the disease.

It is now known that the house-fly only breeds to a limited extent during the hot months; in the Sinai desert, for example, one could observe the imagines emerging from the pupa cases, and after piercing their way through the sand, being immediately destroyed by the scorching rays of the sun.

It is a well-ascertained fact that the house-fly becomes quite a rare insect during the period from June to August; when climatic conditions become more favourable it again is able to multiply, and it is just at this time bacillary dysentery makes its appearance amongst the troops.

Bacillary dysentery is then essentially a disease of standing camps. The greatest outbreaks occurred after troops had remained stationary for some considerable time, and

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the ground had become contaminated in consequence, leading one to presume that a proportion of the house-flies had become infected with the bacillus in the meantime.

There was little evidence that the disease was communicated by direct contagion through the medium of food or water. The chlorination of water was always so thoroughly carried out, as we knew to our cost, that I fail to see how any organism, especially such a delicate creature as the dysentery bacillus, could survive for any length of time.

By suitable technique I was able to isolate Shiga's bacillus from the intestinal tract of house-flies caught in the month of October in the open desert, about two miles distant from the nearest camp, and far from any human fæcal deposits. From one particular batch of twenty flies, by dissecting out the lower portion of the intestinal tract, I was able to isolate six colonies of Shiga's bacillus; from this I inferred that the infection of these insects must have been a pretty heavy one. Laboratory experiments would indicate that the bacillus is capable of surviving in the intestinal canal of the house-fly for a period of four days—observations which have recently been confirmed by TAYLOR in Salonika. I am of the opinion that the relationship between this organism and the fly is a more intimate one than is generally suspected. In the first place, the number of Shiga carriers passing purely fæcal motions is an extremely small one, and must form a very small percentage of any community, such as the Egyptian Labour Corps, which might reasonably be regarded as the reservoir of infection. Secondly, I have found that the life of the bacillus when exposed to the direct rays of the sun in the open desert is an extremely short one—at the most one of four hours. Therefore the fly must ingest a large number of bacilli directly the dysenteric motion is passed to be in position to spread the epidemic in the community, and, moreover, it must have the opportunity of feeding upon blood and mucus stools, which, under the sanitary conditions so happily prevailing, were not to be found lying about in large numbers in the vicinity of any camp I have ever been in.

An observation I was able to make was that the bacillary infection appears to cling to old camping sites, and after a short incubation period would make its appearance amongst new troops settling down in the same area; on the other hand, cases of amœbic dysentery began to crop up some six to eight weeks after an advance into new territory, and while the troops were still on the move.

ETIOLOGY.

As regards the etiology, I have no hesitation in saying that I am one of those "conservatives" who still consider that bacillary dysentery is caused by two organisms only: Shiga's and Flexner's bacillus—call it Flexner-Y if it is preferred—but I most emphatically do not believe that an epidemic disease of this description, with such characteristic symptoms and pathology, is caused by a score of organisms whose sole claim to pathogenicity lies in the fact that they can be cultured from dysenteric fæces, and when placed in solution of carbohydrates, give reactions somewhat similar to those of the well-recognised dysentery germs. It seems to me that the natural inclination which microbiologists seem to possess for multiplying pathogenic bacteria, often upon very insufficient evidence, *ad infinitum*, is a very dangerous one to follow—a craze, I might mention, not confined to bacteriology—and I think we should have great hesitation in accepting any subvarieties of Shiga's bacillus. I regard that bacillus as one and indivisible. It is surely not justifiable to regard an organism which happens to be isolated from a dysenteric evacuation as the cause of the disease. I therefore entertain considerable hesitation in accepting new varieties of this bacillus, as, for instance, the organisms known as SCHMITZ'S bacillus, D'HERELLE'S bacillus, the *Bacillus ambiguus* of ANDREWES, or the paraShiga + or paraShiga – of DUDGEON. If etiological agents at all, they must play a very minor part, and it is my experience that organisms giving similar biochemical reactions to these may be isolated from true Shiga stools which have been allowed to decompose for a few hours. I am therefore of the opinion that they may be either concomitants, the products of a stale dysentery stool, or that they are derived from the necrotic mucosa. Further research is required before the matter can be considered to be definitely settled.

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As regards the Flexner-Y group, I consider them as having in their different strains a greater range of antigenic power. They may be sub-divided upon agglutination reactions into five subgroups, but I do not conjecture that any hard and fast line can be drawn between each group. It will be found that the Flexner bacillus is not such a well-defined species as the Shiga organism. Any classification of the former bacillus by means of its sugar reactions I regard as unsound, and only two of these remain constant after frequent subculture, namely, those with glucose and mannite; the remainder, especially saccharose and maltose, may be made to vary by subjecting the same strain of bacillus to different conditions.

According to my experience there is no special prevalence of the two main types of bacilli at any particular season. While engaged in doing this work, from 1915 to 1919, I found that the types of organisms isolated from acute dysenteric stools were about equally divided.

I regard the Shiga bacillus as much the more toxic of the two; all the really severe cases of bacillary dysentery, from a clinical point of view, were due to this organism. A Flexner-Y infection may occasionally prove fatal. I have seen one British case, out of twenty post-mortems, in which the organisms were isolated from the intestines. On the other hand, this bacillus is more frequently found in the chronic stage of the disease (14 post-mortems:—Shiga's bacillus, 6; Flexner-Y, 8).

Pathology.

A great variety of the pathological picture is seen, according to the severity and duration of the disease:—

(*a*) *Mild Cases.*—The earliest lesions of bacillary dysentery which have come under my notice consist of superficial "snail track" ulcers, spreading along the free edges of the *valvulæ conniventes*, and originating apparently in the lymphoid follicles of the large gut.

(*b*) *Very Acute Cases.*—Should life last long enough, the fully developed pathological picture consists of a colliquative necrosis of the mucosa of the whole of the large intestine, generally as well as that of the last few feet of the ileum. The lesions are most marked in the lower part of the canal.

The earlier stages of this process appear to consist of the most intense inflammation of the mucosa, which is very friable and bright red in colour; signs are present of a widespread toxic absorption; thus there may be a general lymphoid peritonitis, with free serous fluid in the peritoneal cavity, and œdema of the mesentery at its posterior attachment. The mesenteric glands may be enlarged, hyperæmic, and softened. In the stage of necrosis the mucous membrane of the large gut is greatly thickened, and is converted into an olive-green, or it may be a blackish substance, one-eighth inch in breadth, which is hard to the touch, and which represents the necrotic mucosa.

Should the patient survive, as he seldom does, such an extensive destruction of the bowel wall, the defunct mucosa is exfoliated in much the same manner as a diphtheritic membrane, leaving behind a raw, bleeding, granulating surface.

In a bowel which has undergone such disintegration, complete regeneration of the mucosa does not take place; such restoration of the mucous membrane proceeds from islands of mucous membrane which escape unscathed. The whole process of repair would appear to constitute a struggle between proliferation of the specialised epithelium and fibrosis.

Chronic ulceration of the large bowel in bacillary dysentery takes place in varying degrees of severity. The smallest lesions consist of lenticular ulcerations of the mucous membrane, involving the mucosa alone; the more advanced, of ulceration of limited tracts of the mucosa, rarely penetrating beneath the muscularis; that ulceration may very rarely proceed to ante-mortem perforation appears beyond doubt. In a series of 300 post-mortems I have seen this occur twice.

The ulcers are generally roughly ovoid in shape, and run transversely to the long axis of the gut.

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The following are points by which they may be distinguished from amœbic lesions:—

(1) The ulcers commence on the free edge of the *valvulæ* and run transverse, not longitudinal to the long axis.

(2) Then in shape they are irregular in outline, with ragged undermined edges, often intercommunicating with neighbouring ones; in contradistinction to the oval and rather regular shaped, isolated amœbic ulcer.

(3) The intervening mucous membrane is hyperæmic, œdematous and plum-coloured, and there is no compensatory thickening of the gut; in amœbic ulcers, on the other hand, the intervening mucous membrane is healthy and there is considerable hypertrophy of the gut wall.

Another pathological condition, the direct sequela of chronic bacillary dysentery, which has attracted little attention, are tapioca-like mucus retention cysts, varying from microscopic proportions to the size of a cherry stone, which jut out on to the mucosa, and are situated beneath the scars of old ulcers. These cysts become secondarily invaded by *B. coli* organisms and frequently a peculiar *B. coli* septicæmia results, which ENRIGHT and myself have described, leading to formation of pyæmic abcesses in the cortex of the kidneys and very often to a fatal termination.

The mucus retention cysts appear to be formed as the result of an adenomatous downgrowth of LIEBERKÜHN'S follicles into the submucosa; apparently in the formation of scar-tissue part of the fundi of the crypts are nipped off.

Polypoid outgrowths reaching ¾ to 1 inch in length, scattered throughout the rectum, have been observed as the result of a chronic bacillary infection.

I have isolated the specific and typical dysentery bacilli from the gut in all stages of the disease. When the mucous membrane is necrotic, it is necessary to remember that successful isolation depends upon procuring material from *beneath* the necrotic tissue, where alone the bacillus can be found; for this purpose one should sear the tissue with a red-hot knife, and then scrape it away. Failure to remember this results in the isolation of putrefactive organisms, such as *B. pyocyaneus*, which are found in necrotic tissue and which I am convinced have nothing whatever to do with the pathogenesis of acute dysentery.

I have never succeeded in cultivating the bacillus from the bile or blood, though LEDINGHAM, and more latterly BOYD (*Lancet*, Sept. 13th, 1919), have recorded successful isolations of Flexner-Y bacilli during life.

SYMPTOMS.

It may safely be said that the clinical symptoms of bacillary dysentery are never so characteristic that the clinician can afford to neglect the advantages of a laboratory diagnosis, and there is no disease in which the mutual co-operation of the clinician and pathologist are so necessary.

All degrees of severity may occur; I have seen a mild diarrhœa of three days' duration with passage of a blood and mucus stool, from which I isolated Flexner-Y; other cases in which, with the minimum amount of discomfort, one or two solid motions, with a coating of gelatinous blood and mucus, proving to be a Shiga infection, were passed. Again, I have seen many fulminating or choleraic cases, in which death occurred within three days from the onset, and one, especially I remember, in which death occurred in as short a period as fifty-six hours.

On clinical grounds, bacillary dysentery can be classified into five types—(*a*) the mild, (*b*) acute, (*c*) toxic, or fulminating, (*d*) relapsing, and (*e*) chronic.

The *fulminating type* may be divided into two subgroups—the choleraic and the gangrenous. The first of these is somewhat rare, but may bear a close resemblance to cholera. The onset is acute, generally with vomiting; collapse with its attendant phenomena set in early. The temperature is subnormal; the tongue dry and glazed; the skin cold and clammy, and the patient may complain of cramps. There is an initial watery diarrhœa, which is soon replaced by dark-red mucus, containing a high proportion of blood, or, it may be, serum alone.

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The gangrenous form also commences suddenly with a rigor, headache and vomiting, and other evidences of a severe toxæmia. The face is flushed; the pulse rapid and bounding. The abdominal pain and tenesmus are very severe, but as the toxæmia increases these wear off. This is a point in prognosis: beware of cases with pyrexia in those who become insensitive to abdominal pain; it is by no means a favourable omen.

The stools at first resemble "meat washings," but towards the end are composed of dark-grey offensive fluid, containing much altered blood. The underlying pathological cause of the absence of mucus is to be found in the total destruction of the goblet cells which has taken place.

An important point to remember about the chronic form, which is more frequently seen, fortunately, in debilitated natives, is its intractability, and the nature of the stools, which may shew no external signs of blood or mucus for many months at a time; add to this the great difficulty, amounting sometimes to an impossibility, of isolating a dysentery bacillus from the fæces (*though it may be present in the intestinal wall and can be found at autopsy*) renders the diagnosis of these cases during life a matter of very great difficulty indeed.

Of complications, conjunctivitis, iritis, and polyarthritis have been met with; the latter may resemble that of rheumatoid arthritis. A unilateral parotitis has been observed.

Diagnosis.

In making the diagnosis of bacillary dysentery it may be laid down that the pathologist himself should have had a considerable experience of the work, and should be alive to the rather complicated changes which are proceeding in the intestinal canal of the patient.

As regards any information to be obtained from the stools, though they alter pretty considerably during different stages of the disease, nevertheless in early cases, I think, we can lay down certain rules which may serve as an index to the clinician, though I admit there are exceptions. The acute bacillary stool consists of pure blood and mucus—or, better still, "bloody mucus"—it is in fact, mucus tinged with bright red blood, of extreme viscosity, and tending to adhere to the bottom of the bedpan or containing vessel. It is odourless, or bears a faint smell of spermin. It, in fact, represents an acute inflammatory exudate, derived from the mucosa of the whole or major part of the large intestine. The amœbic stool, from which it is necessary to differentiate it, is composed of blood and fæces intimately mingled, is very offensive, not viscid, and represents the exudate and sloughs derived from the ulcers throughout the canal, the dark altered blood being derived from small intermittent hæmorrhages at the bases of these ulcers.

The diagnosis of bacillary dysentery can be made sufficiently accurate for all practical purposes, by a straightforward examination of the cellular exudate alone. In military practice, what is most required is *promptness;* it is necessary to diagnose early *in order to save the patient's life;* the clinician in a casualty clearing station cannot afford to wait twenty-four hours before applying the appropriate remedy. *If a few hours' delay takes place, it may result in irreparable damage to the gut-wall,* and one cannot restore a once-scarred and fibrosed intestine.

The most characteristic feature of the bacillary stool, as seen under the microscope, is the large number of undamaged polymorphonuclear leucocytes; excluding red blood corpuscles they constitute quite 90 per cent. of all the cells. In addition there are large cells, the macrophages, 20 to 30μ in diameter, very characteristic cells in the *early stage* of the disease, and apparently derived from the capillary endothelium of the submucosa.

They are round, oval, or even bilobed, and may contain vacuoles and granules of various kinds, and even ingested red cells or leucocytes; they are non-motile, and are liable to be mistaken for *Entamœba histolytica*, to which this cell bears a rough resemblance. I have no hesitation in asserting here that these cells are responsible for many of the outbreaks of "amœbic dysentery" in this war. Up to the time of the Armistice they were a constant thorn in one's flesh; every new pathologist who came out was taken in by them till they had made a closer acquaintance with the real amœba, and I hardly like to think

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of the amount of emetine which has needlessly been wasted as a consequence, with the idea of combating an imaginary amœbic infection, and to the detriment of the bacillary cases.

Entamœba coli may occur also in acute bacillary stools. I have seen a Shiga stool which was swarming with these protozoa. These organisms also contribute a source of difficulty and confusion, and may be responsible for a number of double amœbic and bacillary injections which have been notified. On the other hand, in the majority of cases, the amœbic exudate contains very scanty polymorph cells, very few macrophages, and large numbers of red cells in rouleaux. Of course I recognise there are exceptions, and that the microscopic appearances of the bacillary exudate varies very much during different stages of the disease.

Isolation of the bacillus is a simple matter if the stool is obtained fresh from the patient early in the course of the disease and uncontaminated with urine. The mucus which, if contaminated by fæces, should be washed in sterile saline, should be spread thickly in a spiral manner on a McConkey plate, or, better still, litmus-lactose agar; it is better not to have any inhibiting agent, such as taurocholate in the former medium.

The plate should be incubated for eighteen hours at 37° C., and then examined for the characteristic colonies by means of a watchmaker's lens.

The dysentery colonies hardly ever constitute the majority on the plate (I have only once seen a plate in which they constituted 80 per cent.); more generally they are of slower growth than other bacilli, and are rapidly inhibited by *B. acidi lactici*, *B. coli*, and especially by *B. pyocyaneus*.

For the sake of speed and accuracy in diagnosis, I prefer the method of preliminary macroscopic agglutination on Garrow's agglutinometer, which is utilised as follows:—

As many colonies as possible are picked off from the plate by means of a platinum spud and emulsified in a small quantity (·25 c.c.) of saline till the fluid is distinctly opalescent; the emulsions are then mingled by means of a drop-pipette on the glass slab with an equal quantity of specific anti-Shiga or anti-Flexner-Y serum in a dilution of 1:50, giving a resulting dilution of 1:100. After rotation of the slab for three minutes, characteristic macroscopic agglutination becomes very evident. The bacilli may then be confirmed by agglutination in still higher dilutions, if so desired.

It has been my experience that this constitutes a very valuable, accurate and rapid means of diagnosis.

The emulsion which remains can then be placed in solutions of the various sugars, and, in my experience, this invariably confirms the results given by the macroscopic agglutination test.

The Widal Test as a Means of Diagnosis.—As regards the value to be attached to the Widal test, as applied to the diagnosis of bacillary dysentery, I have little to add to what has already been written. The test, if it is to be of any value, must be performed by the macroscopic method and with standard and sensitive strains of the organisms. With Shiga's bacillus the results seem to be accurate, if agglutination in a dilution of over 1:25 is taken as a standard. The Flexner-Y bacilli on the other hand, are much more liable to be clumped by normal sera, and this reaction must be eliminated. I would therefore consider nothing under a dilution of 1:100 as being of any value whatsoever. Considering that the agglutinins do not make their appearance till the patient is almost convalescent, one can place little reliance upon this serological reaction as a means of diagnosis in the acute stage of the disease.

Treatment.

The watchword in the treatment of bacillary dysentery is, "early diagnosis, early and vigorous treatment"; more especially is this true in the acute types of the disease.

The main principles consist of placing the patient so that his intestinal canal is at rest and that the diet he absorbs is as nutritious as possible, with the least amount of non-absorbable residue.

The treatment of fulminating cases.—These require the most vigorous measures,

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and the methods adopted may be applied with modifications to the less acute clinical forms.

If collapse is imminent it is best to keep the patient warm with hot-water bottles. In my opinion, when the passage of stools is almost continuous, the patient should, on no account, be permitted to exhaust his strength by straining on a bedpan; it is much better in these cases to pack him well with tow or cotton wool on a waterproof sheet, which can be changed every few minutes.

Drugs.—It is customary to commence treatment by a preliminary purge in order to clear the large intestine of any remaining fæcal contents; the best for this purpose is ½-ounce of castor oil, containing about 15 minims of *tinct. opii.*

The following morning, the routine treatment with saline aperients should be commenced. The best salt is the sodium sulphate, and should be given in drachm doses, every two hours for the first twenty-four; thereafter, four-hourly, until the stools become fæculent. The routine use of opium in the treatment of bacillary dysentery cannot be too strongly deprecated. Its main uses, and I maintain that it has them, are to procure rest and sleep, and it may be given occasionally when necessary.

The routine use of bismuth has many adherents, but it has little advantage save to clog up the bowel, which should be kept clear of contents as much as possible.

In Central Europe, the exhibition of *bolus alba-kaolin*, with the addition of animal charcoal, in doses of three tablespoonfuls of each, has come into prominence. It is said to check excessive diarrhœa and act as an intestinal antiseptic.

The exhibition of intestinal antiseptics, such as salol or cyllin, do not appear to have much effect in the acute, though they have their usage in the chronic forms.

Treatment by antiserum.—The serum which is placed on the market is a polyvalent one, and, as pointed out by DUDGEON, its chief defect is its deficiency in anti-Shiga immune bodies. If it were possible to obtain a greater degree of antigenic power than is at present the case, I have no doubt that the diversity of opinion which exists as to the value of the remedy would soon disappear.

The main point with reference to antiserum is that it should be given early, and in sufficient quantities; one must remember the pathology of the disease. It would be advisable to bear this in mind when issuing the serum to medical units. The view prevailed till quite late in the war that the proper place to inject serum and allied substance was in the base hospitals, and its issue was not permitted to field units. I maintain that the *field ambulance* is the proper place to give anti-dysenteric serum. In very acute cases, I am of the opinion that the *intravenous* route, in doses of 60 c.c., is the most efficacious.

The next most efficacious method is the *intramuscular* route; this proved to be a most convenient method. A large amount of serum, up to 120 c.c., can be injected with the minimum of pain into the adductor muscles of the thigh, where the intramuscular planes will accommodate large quantities. When it is necessary to give a very large dose, half the amount may be given into each side; care should be taken, of course, to miss the femoral artery. This is a much less painful method than injecting into the flanks or superficial tissues of the abdomen, and, to my mind, much more efficacious.

Anaphylaxis.—There is a danger—a very remote one, it is true—of anaphylactic shock supervening, especially after intravenous injection. I have seen this most alarming phenomenon occur twice—in neither instance was it fatal. Before giving an intravenous injection of any magnitude, it would be well to enquire whether a dose of serum has been given some time previously.

The action of anti-dysenteric is to be regarded as analogous to that of anti-diphtheritic serum (with diphtheria, bacillary dysentery has many points in common), and it must be remembered that once the mucous membrane of the gut has become necrotic, it is doubtful whether the serum has any effect at all.

A certain mild reaction is apt to follow the injection of serum. I have noted that on the day following an exacerbation of the symptoms may even take place, but these soon abate. Should toxæmic symptoms still persist, I believe that injections of the serum should be repeated at an interval of three days.

Serum sickness is apt to supervene six to ten days after the injection; this was specially

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the case when certain batches of serum were used. With others I noticed no reaction whatsoever. I suggest that possibly toxic bodies in the horse serum may be developed as a result of long storage. It cannot be too sufficiently impressed upon officers in charge of base medical stores that all sera should be kept *on ice*, and stores in the field should be frequently renewed.

Diet.

It is quite unnecessary to regard milk as the one and only diet in intestinal disease; as a matter of experience, plain milk, whether boiled or unsterilised, is badly borne by severe cases of bacillary dysentery, besides being unpalatable and monotonous. The casein is passed quite undigested, and appears as clots in the stool.

The ideal diet should be definitely unirritating, easily digestible, and should be as valuable as possible from a nutritive point of view.

A scale of diet, consisting of small feeds given every two and a half hours, is the one I prefer. It should consist of tea, albumen water, jellies, bovril, Brand's essence, and chicken tea, in six to ten oz. doses.

The too rapid introduction of solids into the diet may lead to sudden collapse, or to a profuse diarrhœa supervening in a case which was doing well. On the other hand, it is unnecessary to go to the other extreme and adhere to a too rigid diet of albumen water for a week or more at a time.

As useful additions to the diet may be mentioned boiled arrowroot, cornflour shape and stewed fruit.

As regards local treatment, the mouth should be kept clean. The abdominal pain is best relieved by hot-water bottles or turpentine stupes.

Tenesmus and dysuria are best relieved by a cocaine suppository; if excessive, a morphia injection is permissible.

Treatment of Special Symptoms.

Collapse.—It is most necessary to forestall, if possible, the advent of collapse When once this condition has been established, it is too late to restore the patient. Should this occur, all the usual remedies must be adopted.

Intravenous injections of normal salines should be generously given, up to two or three pints, or even more, as has been advocated. The injection of smaller quantities is followed by a temporary improvement only. To the saline solution may be added water one pint and atropine gr. $\frac{1}{100}$.

I have obtained good effects by intravenous injection of Rogers' hypertonic saline (sodi. chlor. grs. 120, calc. chlor. grs. 4, pot. chlor. grs. 6, water 1 pint, to which may be added glucose grs. 35).

These solutions should be given slowly, and at a temperature of 104° F.

Vomiting and hiccough should both be regarded as symptoms of serious import, and personally I have never seen a case recover in whom the latter symptom made its appearance.

The treatment of the less acute cases should, more or less, follow the lines I have already laid down. Personally I consider the indications for the administration of anti-serum should be based upon the following points:—

(1) If the patient shews signs of toxic absorption.
(2) The stools number more than 18 in the 24 hours.
(3) A remittent pyrexia and a rapid pulse, and great abdominal pain.

The treatment of chronic bacillary dysentery is at the best unsatisfactory, and taxes all the resources and ingenuity of the physician. The best course of treatment, to my mind, is rectal lavage with silver salts, of which protargol, ·5 to 1 per cent, is the most preferable. A well-lubricated stout rectal tube should be inserted, and solutions given by means of a funnel by gravitation, the patient being in the knee-elbow position. Rectal lavage should be combined with abdominal massage, and small doses of salts given by the mouth.

Vaccine treatment has been disappointing.

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I have seen little of appendicostomy in this condition—an operation performed with a view of washing out the large intestine daily—and what I have seen has by no means been favourable.

Finally, for military purposes, I would suggest the following rules for diagnosis and treatment of bacillary dysentery :—

(1) Blood and mucus in the stools of a sudden onset generally mean bacillary dysentery, and the exudate should be reported on, at the earliest possible moment, by a *competent* pathologist.

(2) If immediate microscopic diagnosis is impossible, and the case is severely ill, a moderate dose of 40 to 60 c.c. anti-dysenteric serum should be given.

(3) Whatever the type of case, treatment by antiserum is only slightly more important than treatment by rest and diet.

(4) A diagnosis *once made* must be recorded *legibly* upon the field card or bed-sheet, and the treatment once instituted should be adhered to as much as possible.

By this I mean that a case, once diagnosed bacillary, should continue to have anti-bacillary treatment, and should not be switched off, as so often happens for no apparent reason, on to emetine.

Should *Entamœba histolytica* be subsequently discovered in the stool, this latter course is a rational one to take. I quite admit that a number of cases diagnosed as bacillary dysentery, in the first instance, developed amœbic dysentery at a later date; this apparent anomaly may be explained by the breach of surface which has taken place during the bacillary infection, which has afforded the entamœba a portal of entry to the submucosa of the gut.

Discussion.

The President: We are very much indebted to Dr. Manson-Bahr for the great trouble he has taken in bringing this subject of bacillary dysentery before us to-night. I congratulate him on the able way in which he has dealt with the subject in all its aspects, and I am sure the discussion which will follow will be interesting and instructive.

I shall confine my own remarks to the epidemiological aspect, though I am much interested in the other aspects, which I had the opportunity of studying, along with Sir David Bruce and Colonel Notter, in the Boer War, in the hospitals and camps of South Africa, and in the laboratory which we established in Pretoria.

The author has found living Shiga's bacilli in the intestinal tract of the fly five days after it was infected, also in the intestinal tract of flies which were caught in the open desert. These observations I regard as extremely important, and, no doubt, they emphasise the importance of the house-fly as a carrier of dysentery, and, therefore, the need for eliminating it as an influential factor in the dissemination of bacillary dysentery. On the other hand, I think I must join issue, except in certain circumstances, with Dr. Manson-Bahr's statement that the main factor in the spread of bacillary dysentery is the house-fly. No doubt under the special circumstances which prevailed at Gallipoli, also in the locality where Dr. Manson-Bahr was working, and in Bloemfontein and some other localities in the Boer War, the house-fly played a relatively very important part in spreading bacillary dysentery; but I am certain that in some localities in South Africa the spread of the disease could not be attributed to the house-fly. We are now living in an insect-carrying age, and there is a danger of forgetting that there are other factors besides insects responsible for the spread of disease. We must bear in mind some of the observations of the past. Shiga himself shewed that this particular form of dysentery was

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spread from certain wells in Japan, and that it could be water-borne. He instanced the case of a village whose inhabitants had been forbidden to use a certain river for swimming and fishing because of fish breeding in it. When that prohibition had been removed and people were again allowed to bathe there, a large outbreak of dysentery occurred. It was directly traced to the washing of infected clothes in a village higher up the river. Even in the memorable investigations made in the Spanish-American War there were only 15 per cent. to 20 per cent. found to be due to the house-fly, in the case of typhoid fever, which left other causes to account for the remaining 80 per cent. of cases.

I agree with Dr. MANSON-BAHR that in long-standing camps there is always a danger of dysentery; but much of this may also be due to certain pollutions of the soil and to contact, the infective material adhering to boots, clothes, fingers, and so on.

These remarks are made rather as a warning than from any spirit of criticism of the paper, which is an admirable one in every respect.

Professor ALDO CASTELLANI: I would like, in the first place, to congratulate Dr. MANSON-BAHR on his most interesting address.

I quite agree with him that in the great majority of cases of bacterial dysentery the bacilli isolated may be classified either as Shiga or Flexner. I also agree with him that a number of organisms recently manifested in association with the disease have nothing to do with it. I must say, however, that I have occasionally come across cases from which I have isolated bacilli which cannot be classified as either Shiga or Flexner.

I would like to direct your attention to a bacillus which was described by me many years ago, and which I more recently isolated in a few cases in the Balkanic war zone and at Taranto, and which I have called *B. metadysentericus*. This is, as regards many characters, a dysentery bacillus. It is non-motile, it does not stain by Gram, it does not liquefy gelatine or serum, it does not clot milk, it does not produce gas in any of the carbohydrates or sugars. After a few hours it produces slight acidity in glucose, levulose, occasionally in galactose, and very rarely in mannite. A very important point, to which I would like to call your attention, is that, after ten, fifteen or twenty days, this germ produces a slight acidity in lactose (never gas). The acidity is very slight, but is definable. This germ produces indol, it is non-toxic to the rabbit, it is not agglutinated by Shiga serum nor by Flexner-Y serum, but it is agglutinated by a homologous serum. The blood of the patients from whom I isolated this germ contained, after ten or twelve days from the onset of the disease, agglutinins for this bacillus; no agglutinins for Shiga or Flexner. Therefore I came to the conclusion that there were cases of dysentery, very few in number, due to this germ. At any rate, it seems to me, there are as many reasons for this germ being the cause of certain cases of dysentery, as for the Flexner germ. I still have some cultures of this micro-organism, and I shall be pleased to supply bacteriologists interested in the subject with them. There are several varieties of this organism, which can be easily distinguished by using my absorption method; and, for practical purposes, I advise the modification of the method, as carried out by Colonel CUMMINS and Major ALCOCK.

In connection with the subject we are discussing to-night, I would like to say a few words about another germ (*Vibriothrix zeylanica*, CAST.), which I have often isolated from cases of bacterial dysentery, a germ which, in my opinion, has nothing to do with dysentery, but is probably a noso-parasite, and it is generally found in cases of true Shiga and Flexner dysentery. This germ is most peculiar; the same preparation will shew bacillary forms and pseudo-spirillar forms. It is motile, and its motility is due to the presence of a large number of cilia, which have been discovered recently by ANIGSTEIN in Poland. This germ does not coagulate milk, does not liquefy gelatine or serum, and does not produce gas in any of the known carbohydrates and sugars. On the contrary, there is a distinct production of alkalinity. I do not believe this germ has anything to do with dysentery, but it is often found in cases of bacillary dysentery in which the true Shiga bacillus is isolated. This organism has been thoroughly investigated by Dr. TAYLOR. I call your attention to this organism because it is important from the practical point of view, for on coloured media its colonies are practically identical with those of the true

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Shiga or Flexner. I have brought for your inspection two plates, and, I think, you will agree as to the similarity in appearance to dysentery germs.

In conclusion, I would like to say a brief word about the treatment of bacillary dysentery. On this I am in complete agreement with Dr. MANSON-BAHR. It is the greatest mistake, as a general rule, to give milk as the principal article of diet to these patients. And, as regards drugs, I am very much against giving opium in these cases. The best treatment is to give the sodium sulphate mixture, and anti-dysentery serum in very large quantities.

Colonel S. LYLE CUMMINS: I would like to make a few remarks on this subject from the Army side, but I cannot begin without expressing to Major MANSON-BAHR my great admiration for his paper. I also think that his pathological pictures, which he did himself, are most informing, and give one an idea of dysentery, which, to me, is of great use, and will probably help everyone.

Major MANSON-BAHR seems to combine in his person two elements, which must be found in association in the near future if we are to have good clinical work, namely, great laboratory experience with a keen interest in the clinical and ward side of the work. It seems to me that a person who is only clinical will not nowadays succeed in regard to these intricate tropical diseases; nor will he who is only a laboratory man be able to give all the assistance required. The combination I have referred to must be produced in our schools in the future

Turning to the epidemiological side—on which I can speak more freely than on the bacteriological, because during the war I was engaged chiefly in administrative work—the situation in Mesopotamia, Egypt and Salonica was perhaps very different from what the armies had to face in France. In those other countries the picture was complicated by the fact that amœbic dysentery counted, whereas in France we were able to concentrate on bacillary dysentery. In that connection I would ask the author what is his personal opinion as to the extent to which amœbic dysentery counted in military epidemics in the East. I have heard it said that in this war amœbic dysentery does not count as an epidemiological factor, although it is important as a disease which is always with us, and has to be dealt with from the point of view of the patient's interests. The menace to a force is bacillary dysentery. I am subject to Dr. MANSON-BAHR'S correction in regard to this if he disagrees with me.

Dr. MANSON-BAHR: I quite agree.

Colonel S. LYLE CUMMINS: Assuming that bacillary dysentery was the important disease, the measures we took in France may be of interest in other theatres. In the summer of 1918 we knew at headquarters that dysentery would come—it had come the previous year in July—and although there was none in the forces during the spring and early summer we knew we had to expect it, and we took our measures in advance. The administrative measures were on these lines. We considered the real danger to be trench sanitation. In trench warfare, as in moving warfare, the sanitation of the front line is always bad. The best sanitary officers cannot deal, under machine-gun fire and trench-mortar fire, with sanitation in a completely satisfactory manner. Men are killed while using the latrines, and when going to and from them, and sanitation suffers in consequence. I have never gone round the trenches in active parts of the line without seeing sights which horrified me, and which I did my best to put right, but they could never be put entirely right. Whether due to flies or to the dust, there is no doubt that infection is close by us in the trenches, and it passes from man to man with great rapidity. You must get your diarrhœa cases out of the line, because, when bacillary dysentery is rife, diarrhœa due to dysentery bacilli is the greatest source of danger, the cases of it escaping isolation. We should take these cases to places where they are less dangerous, and where we can examine them more closely and weed them out. The commanding officer will oppose your attempts at removing these cases. It is hard to lose soldiers because they have diarrhœa, and yet it is in the commanding officer's own interests to get rid of them

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for a few days, if necessary. They must be got down to the field ambulance; you do not want to lose to the division any men who can be kept in it. In the field ambulance the patients are still *in the division*, and able to rejoin the units at once if found fit, so field ambulance diarrhœa centres are the things to aim at in the first instance.

How can one get these men bacteriologically examined? We cannot have dysentery centres everywhere. We were able to arrange one or sometimes two per army in selected casualty-clearing stations. At the field ambulances the alternative we adopted was to examine dysentery swabs, instead of stools. That would be futile for amœbic dysentery, but when it is a question of the diagnosis of bacillary dysentery, twenty or thirty "swabs" can be carried by one man in closed tubes to the dysentery centre, fifteen miles away if necessary. This, with a motor cycle, can be done inside an hour, so the time lost is not great. We had one or more dysentery centres in each army. A patient with diarrhœa *who shewed blood and mucus in his stools* would be at once sent down, without further bacteriological examination on the spot. But even when not passing blood and mucus he might be a case of bacillary dysentery, and an agent in the spread of the disease. Therefore, we arranged that such cases as had not the characteristic clinical signs of dysentery should have dysentery-swabs sent to the dysentery centre as already described. In the Second Army, where the matter was taken up energetically, the D.M.S. sent competent bacteriologists to work in several of the field ambulances, and they took an equipment which had no other purpose than the examination of stools—a light equipment which answered very well. I can imagine with what wrath my arrangements must have been received by the officers in charge of mobile laboratories on whom the real strain fell. They examined hundreds of swabs a day, and the strain was tremendous. We got a large proportion of the early cases of dysentery out of the line, and, considering that foci of the disease were present in every part of the line at the same time, I think we got off lightly with 8,000 cases during the summer in an army of over a million men. And every effort was made to save men in France, and not let them go to England, for that meant they would be a long time away. The situation in 1918 in regard to man-power was acute, and we tried to improvise special arrangements for dysentery cases, so that men who appeared to have recovered from the disease could be sent back to the line after further examination. There is a great strain on convalescent depots in war, and it was hard to ensure that the recovering men were adequately supervised. Our full scheme, however was, to some extent, defeated; the fighting was too heavy; still, we saved many thousand men to the force by what we did do. I can see in this room one of the officers who helped to bear the burden put on base laboratories by the arrangements made. The officers who were bacteriologists in isolation hospitals at the base had a dreadful time; there was such an enormous number of cases to deal with. The system must be admitted to have broken down to some extent. It ended in men having to be sent to England who might conceivably have recovered in France. Many were lost, but many were saved. I think work on these lines must be carried out in future cases; you need to get men out of the line when they are in a condition which is dangerous to others, and yet send them back to the line as soon as you are satisfied they have not got dysentery, That can only be accomplished by having a system of dysentery swabs which can be sent to and examined at advanced dysentery centres. And as Dr. MANSON-BAHR says, they must be examined by competent bacteriologists. I gladly accepted a suggestion which Lieut.-Col. C. J. MARTIN made at the beginning of the epidemic, namely, that it is not necessary to examine every case. The bacteriologist will fail if he has too much to do. The important thing is to be sure what type of disease you are dealing with, and to make your arrangements accordingly. A few individual cases may perhaps suffer, but in the main, on the average, you will be right. We found in France that ten per cent. of the cases were Flexner, and, for the broad purposes of administration, the outbreak was a Flexner epidemic. Now, the examination of ten per cent. of stools would be sufficient to establish that fact. But the bacteriological investigation must be deliberate; any hurry is a mistake; one has, after all, to cut one's coat according to one's cloth. At a later stage there must arise the question of the treatment of the individual. If, in any given epidemic, you adopted the plan of giving a

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monovalent serum, you might be losing men. Your samples will give you the necessary information, and then you will use a mixed or a monovalent serum, as the bacteriological findings indicate.

The question of amœbic dysentery also comes in, and that is a serious one, because if you are to assume, on sampling, that all the cases are bacillary dysentery, the treatment of the others may be wrong. I think Dr. MANSON-BAHR has put his finger on the right point there: you do not need to carry out elaborate examinations for that. A direct microscopical examination of the stool will give much information even when amœbæ cannot be found. One can nearly always tell, from the cellular content of the slide, whether it is the amœbic or the dysenteric form.

I would like to ask Dr. MANSON-BAHR, merely from the point of view of arrangements in a future army on service, what he considers the best form for provision of anti-dysenteric serum. Should the supplies include monovalent serum for Flexner and Shiga? I assume that one must always have a mixed serum for use at a period when the diagnosis is still uncertain: I mean, rather, for subsequent treatment. And how should the serum be sent? A 10 c.c. phial is convenient in many ways, but it is, I think, most inconvenient to the man who wants to give a large volume, say, 60 c.c. to a case.

I am sure, with the author, that one must give large doses, and in the end it might be better to have a larger phial sent out. Definite recommendations by people who have been working in casualty-clearing stations during the war would be valuable, and might be of help in the proper quarter, especially if made at such discussions as this.

With regard to means of transmission of the disease, I think Dr. MANSON-BAHR's work on the fly is of enormous value, and finally settles a point which formerly was much in doubt. In his paper he said drying killed these bacilli, and I ask whether he worked with cultures or mucus in his experiments. I have an impression—and I have some grounds for it—that dysentery bacilli will stand drying, at any rate desiccation, to a much greater degree than is generally believed.

That brings us to the question of dust infection. Two questions arose in France—two points which puzzled me, and on which I would like information. How does it occur that, in a force of over a million men, extending over a 100-mile front, dysentery develops all along the line, or at points very distant from each other, simultaneously? That is what happened in France in 1918, for within a week we went from no dysentery to a large number of cases. They were practically all Flexner. There had been a lot of flies with us during the preceding month, though they were never completely "out of hand," as they often are in the East.

The other question which baffled me was, Why, while dysentery in Eastern theatres of war was very severe, in France was it so mild? The number of men in France who developed the sort of intestine of which we have seen pictures to-night were very few. The cases were unable, for a week or two, to take solid food without getting a recurrence of symptoms, but they were not bad cases.

Another question, not so germane to the subject, is, Why is it that, in view of WARRINGTON YORKE's work, shewing that we have a large number of amœbic cyst excreters in Europe, in people who have never been abroad, yet we do not find amœbic dysentery and hepatic abscesses developing in England? I would like to know what physiological factor is behind these problems, as I think there must be a physiological side to the subject. Perhaps one ought to experiment rather than speculate: it occurs to me that the question of suitability of food may come in. People who are living on European diet in Europe can deal with that diet very well. When, however, they go the East and have the same diet, a good deal of portal congestion may perhaps be set up, and that condition of affairs may greatly help the amœbæ, and conduce to a greater severity of the disease. If the influence of such factors were thoroughly investigated, I think we might find a clue as to how to control the severity of the disease.

Dr. H. FRASER: It is possible to exaggerate the importance of the part played by flies in the spread of dysentery, with the result that the simpler and more obvious pre-

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ventive measures are either neglected or ignored. In my experience, dysentery is principally spread by direct infection from the sick to the healthy.

The part, if any, played by flies is a minor one. The finding of dysentery baccilli on the feet or in the gut of flies is interesting, but is not proof that dysentery is spread in this way.

In the diagnosis of the type of dysentery, the assistance of the laboratory is valuable, but the clinician must recognise the extent to which that assistance is of value and not lean entirely on the work of the pathologist.

The diagnosis of the type of dysentery, from the macroscopic or microscopic appearance of the stool, is useful in a general way, but is not reliable. I have seen exactly similar stools from cases of amœbic and bacillary dysentery.

The clinician is sound in regarding every case of dysentery as amœbic when he finds amœbæ, or when the pathologist reports the finding of amœbæ. It is immaterial whether the pathologist regards the amœbæ as *coli* or as *histolytica*. If one or other of the dysentery bacilli are also found, it does not alter the diagnosis, but merely indicates a mixed infection.

For the moment, emetine holds sway in the treatment of amœbic dysentery. Perhaps it does do good, but one remembers the day when ipecacuanha *sine* emetine held the field.

When histolytica cysts and no amœbæ are found in the stools, emetine-bismuth iodine does not cause the cysts to disappear.

The difficulty with the non-amœbic cases is the number that give negative bacteriological findings. I am not dealing with the cases examined in the first few days of the disease when positive findings are the rule, but I am dealing with the cases as a group under the conditions with which we meet them in civil life. When positive findings occur, the results can be communicated to the clinician in from 18 to 24 hours by the method which we have employed for years at Kuala Lumpur. The material is spread on litmus-lactose-agar plates, which are incubated at 37° C. for not less than 18 hours. Two glass slides are taken, on each is placed a series of drops of 1–25 dilution of agglutinating serum of high fibre; the one slide being for Shiga serum, the other slide being for Flexner serum. Touch the likely colonies with a platinum needle and rub up the adherent organisms in one of the drops. The results are visible to the naked eye and are reliable.

There is no necessity to carry out sugar reactions. We prepared our own agglutinating sera and recognised only two types of dysentery bacilli—the mannite-fermenters and the non-mannite-fermenters.

Even with the most skilled assistance, the clinician will still be left with a considerable number of cases in the diagnosis of which the laboratory cannot assist him. Clinically dysentery, he will treat them as such, and in the treatment of non-amœbic dysentery, rest, salines and restricted diet yield as great a percentage of recoveries and as low a mortality as any method which has yet been devised. The serum-treatment of dysentery is not satisfactory. We tried, with some degree of success, specific sensitized vaccines, using anti-dysentery sera prepared from rabbits. This method of treatment involves the determination of the causative organism, and would not be applicable to the group of cases from which the causative organisms cannot be isolated.

Dr. BRITTAN GILL: With great pleasure I have listened to Dr. MANSON-BAHR'S remarks, more especially as I happened to take part in the treatment of the dysentery epidemic in the autumn of 1917, at the *base* in Egypt. I would like to disabuse Dr. MANSON-BAHR'S mind of the idea that a large number of the cases arrived at the base *minus* their history sheets; the majority of those evacuated to No. 17 general hospital had their documents complete.

I will limit my remarks to-night to two questions. The first bears on the diagnosis of bacillary dysentery in a country where malaria is very prevalent. A large number of the cases which came under my notice were complicated ones, for though the cultures were positive for dysentery, the cases did not respond to the serum treatment. Not being satisfied, we took blood films, and found the patients were heavily infected with malarial

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parasites, and intramuscular injection of quinine resulted in the symptoms clearing up. A good deal of work was done on what may be called dysenteric malaria. A lesion occurred in the intestine, similar to that in bacillary dysentery, due to the capillaries being blocked with parasite-laden corpuscles. I would like to hear anything further Dr. MANSON-BAHR has to say on differential diagnosis.

The other point is in regard to arthritic symptoms in dysentery. I would ask whether Dr. MANSON-BAHR has decided that serum can be excluded as a cause of the arthritic symptoms in these cases?

Dr. WILLIAM H. WILLCOX: I would like to express my congratulations to Dr. MANSON-BAHR for the admirable paper he has given us. Everyone who has been among much dysentery during the war must realise, from every word the author said, that he was speaking of what he has seen.

I would like to say just a few words in order to emphasise the importance of the points Dr. MANSON-BAHR has made in regard to the treatment of dysentery. For three years, as consulting physician in Mesopotamia, I had opportunities of seeing enormous numbers of dysentery cases, and it is very true that unless the severe cases of bacillary dysentery are given serum early, the prognosis is rendered much graver. It is doubtful, indeed, if serum does much good in the later stages. In Mesopotamia, especially during 1917-18, when we had the Turks on the run and our troops were advancing over very long distances, we had some very tragic experiences. I remember, especially in the areas around Kirkuk and Kifri, where, during a rapid advance, a large number of severe cases of bacillary dysentery occurred. The high mortality was largely due to the long journeys necessitated before hospital treatment could be obtained and serum given. All possible steps were taken to try and deal with this defect, and the field ambulances were supplied with serum and instructed to use it as early as possible in cases which were clinically bacillary dysentery.

The author did not, I think, allude to certain clinical differences, in the early stages, between amœbic and bacillary dysentery, namely, the furred tongue, the acute onset, the fever, etc., in bacillary dysentery, symptoms which often are absent in the amœbic form, and also the naked-eye differences in the stools. When there is not expert pathological aid at command, those clinical signs prove valuable guides as to the line of treatment to be adopted without delay.

I endorse all the remarks made by Dr. MANSON-BAHR relating to management and treatment, and these especially apply to places like Mesopotamia and Palestine, where transport conditions were often unsuitable for acute cases. Perhaps they do not apply with such force to conditions in France, such as those to which Colonel CUMMINS has alluded. From my war experiences I feel, very strongly, that laboratories and bacteriologists are especially needed in the front areas, as also are consulting physicians and expert advisers generally. It is by this means that outbreaks of disease are recognised at the earliest possible moment when opportunity offers of nipping them in the bud. I also agree with the author as to large doses of serum. In Mesopotamia our practice was to give three doses of 60 c.c. in the first 24 hours, either intramuscularly or subcutaneously; but, if given intravenously, I think one large dose, 80 or even 100 c.c. is sufficient.

I also endorse Dr. MANSON-BAHR'S view in regard to milk as a diet for these patients. Milk I regard as unsuitable, except when diluted or peptonised, and it should not then be given in large quantities. Albumen-water, to which four per cent. glucose or milk sugar or honey, or arrowroot made with water, to which white of egg is added when cool, or sago boiled until it is of slimy consistence with water and sweetened with glucose or milk sugar, form admirable diets for the cases, as well, of course, as soups, broths, and raw eggs beaten up in diluted milk.

Dr. C. M. WENYON: I am sure that most of us here who have been in contact with large outbreaks of bacillary dysentery during the war could talk about the subject for hours. I do not wish to say more than a few words, even though the somewhat startling statements made by Dr. FRASER, of Kuala Lumpur, make me feel that that the discussion

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is becoming rather interesting. Though I do not agree with all Dr. FRASER has said, still I sympathise with him in his remarks about the unsatisfactory nature of anti-dysentery serum in the treatment of this disease. As so often happens, serum fails to cure the disease, and to explain this the excuse is made that the serum was not given early enough. I should like to see a really careful experiment conducted, and I have suggested this to many medical officers who had charge of dysentery wards. What I wanted to see was a large series of cases treated alternately with and without serum. Every alternate case, quite apart from its severity, to be given serum. I wonder what would be the result. No one was daring enough to risk his reputation in such an experiment, and until such a trial is made on a large scale—and now that the war is over I fear the opportunity has gone—we shall not have any very decisive data on the subject.

I quite agree with Dr. MANSON-BAHR that the atypical dysentery strains of bacteria are of little practical importance, though they may be of great interest from the academic point of view. Because such an organism has been isolated from a minute particle of mucus, is there any proof that this has been the causative organism of the disease? I often feel that if more extensive cultures had been made from such cases, or more certainly if the large intestine itself could have been inspected, and cultures made from the mucous surface itself, this atypical strain would have appeared much less frequently. It is certainly true that the earlier cultures are made after the onset of symptoms, the more frequently are the typical Shiga and Flexner strains isolated.

Dr. MANSON-BAHR has explained that he considers the fly the chief cause of spread of bacillary dysentery. Amœbic dysentery, he thinks, is spread by water more than by the fly, because after the advance in Palestine, when the troops were advancing over ground previously occupied by Turks, and of necessity drinking much unprotected water, the amœbic dysentery increased. Bacillary dysentery, according to him, is a disease of standing camps, and amœbic dysentery a disease of the march. We all know that amœbic dysentery is a very chronic disease, and that many men are infected with *E. histolytica* who do not shew any signs of their infection. When such individuals are made to march under very trying circumstances of fatigue and irritating food, it would not be surprising if these factors did not produce symptoms of their infection, which was already present, though not evident when they were living under more tranquil circumstances. The fly fills its gut with fæces, and, within a couple of minutes, it passes out of its posterior end a drop of material big enough to infect not only with bacillary dysentery but also amœbic dysentery. The only point necessary is that the material shall not be dry; and it seems to me that the comparatively large drop deposited by the fly in any moist substance intended for food, is more likely to produce infection than the minute quantities that are carried about on fingers, where these are much more subject to desiccation. I do not say the fly is the only method of spread of dysentery, but it is a very important one. Any factor which will bring about the spread of moist fæces will cause outbreaks of dysentery.

There is still one other point—I quite agree with the lecturer that acute outbreaks of dysentery are of a bacillary nature, and that where this is known to be the case by bacteriological examination it is of great importance to commence the treatment of dysenterics early, even before a bacteriological result can be obtained. For many reasons the latter may be impossible. One cannot have laboratories everywhere. The lecturer said in such cases it would be well to give a dose of 40 to 60 c.c. of serum, though, earlier in his paper, he said that if serum is to be any good it must be given early and in large doses of 60 to 100 c.c. Why give a smaller dose just because bacteriological diagnosis is wanting? If one decides to treat the case as one of bacillary dysentery, by all means treat it properly and not by any half measures.

Major E. E. AUSTEN: I would like to raise one point in regard to the fly-dissemination theory of bacillary dysentery.

Before doing so, having been associated with Major MANSON-BAHR in another sphere, while serving with the E.E.F., perhaps I may be allowed to add my congratulations to those of others on what he has brought forward to-night. If any justification were

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needed for the author's persistence in urging the establishment of mobile laboratories at or near the front, it is surely afforded by the brilliant results just presented to us.

The reputation of the house-fly as a disseminator of disease has been aspersed by at least one speaker this evening. My own recollections, however, of the findings of the medical commission appointed to determine the origin of the outbreaks of typhoid fever in American concentration camps, during the Spanish-American War of 1898, is that it was stated that the epidemics were mainly due to flies, that flies had caused more casualties than the bullets of the enemy, and that both food and water were exonerated as agencies.* Major MANSON-BAHR has told us that while bacillary dysentery is a disease of late summer and early autumn, minor outbreaks may occur during spring. He also says the bacillus will not stand the direct rays of the sun in the desert for more than four hours. In temperate climates, the period of prevalence of the house-fly is late summer and early autumn, but under the hot sun of Southern Palestine this period is much extended. Spring in Egypt, Sinai and Palestine is the season of the *khamsin*, an oppressive hot wind which dries everything up, making articles of all kinds hot to the touch, respiration difficult and life a burden; the air during a *khamsin* feels as though it had come straight from an oven. As everyone knows, flies believe in the precept "Early to bed and early to rise," and ordinarily they retire to rest about sundown. At Deir-el-Belah, in Southern Palestine, May 19th, 1917, when there were many flies, was a day of *khamsin*; at night there was an extraordinary change in the habits and demeanour of flies in tents; the insects were apparently maddened by thirst, and they were extremely active all the time lights were going in the tents, and for long after they had been put out; I well remember that after I had turned in, and until at least 11 p.m., flies were constantly impinging on my face and getting into the corners of my mouth, in the endeavour to procure liquid refreshment. If Major MANSON-BAHR can establish a connection between a dysentery outbreak and a previous *khamsin*, and if dust as an agency can be excluded, it seems to me that the behaviour that I have just described affords strong presumptive evidence for incriminating the house-fly.

Major MARRIAN PERRY: There is one aspect of the diagnosis of bacillary dysentery which Dr. MANSON-BAHR has dealt with very briefly. That is the serological method of diagnosis. Admittedly the isolation of one or other of the dysentery organisms, or failing that, the microscopic character of the cellular exudate, is of the first importance in acute cases. The patients are not, however, always accessible to laboratory examination in the acute stage of the disease, and this particularly applies to dysentery laboratories at bases. Convalescents arrive in large numbers, evacuated from the front line without laboratory tests, and it is necessary to classify them. It is in such cases that the importance of serological diagnosis by means of the agglutination test arises. We have had considerable experience of this method in France, where, in 1918, a great number of cases passed through our hands, and we have formed a high opinion of its value. In cases of Shiga infection the test yielded 100 per cent. of positive results, and, even in the milder Flexner-Y variety of the disease the percentage was almost as high, provided the patient's serum was tested against a range of strains of these organisms. We were able to confirm the agglutination findings in a large series of cases by later isolation of a dysentery organism of the same type as that indicated by the serological test.

Major C. E. F. MOUAT-BIGGS: I consider that the relative frequency of amœbic and bacillary dysentery can only be fairly determined at or near the actual front line, especially in countries like East and West Africa where the conditions of temperature and moisture seem to keep amœbic activity noticeable for a long time, and that if decided by cases

* "Special attention to the rôle played by the fly was given by REED, WARD, and SHAKESPEARE in their studies of the prevalence of typhoid fever in our army camps in 1898. They concluded that flies undoubtedly served as carriers of the infection, and attributed about 15 per cent. of the cases to this mode of communication."—ROSENAU. [*Ed.*]

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away from the front line, *i.e.*, at C.C.S's or farther back and at the base, wrong deductions are made, as then amœbæ have diminished in numbers and are hard to find, while bacillary infections—if secondary or mixed—are still prominent and the cases are returned as bacillary infection only. I believe that at the immediate front in the campaign in East and West Africa the amœbic cases were far more frequent than bacillary, and where bacillary infection took place they were *secondary* and not "mixed" infections purely, and that this was indicated by the characteristic changes from a pure glassy stool (following a fresh purely fæcal stool) which swarmed with active amœbæ, after the second or third day to a milky white opacity, obscuring the amœbæ and shewing characteristic epithelial débris of *secondary* bacillary infection. I remember an original communication in the *Journal of Tropical Medicine*, of March, 15th,, 1919, Vol. XXII., by Captain WEST, M.B., where the author particularly noted these points in his experience in East and West Africa in late campaigns.

Dr. P. MANSON-BAHR: With reference to Dr. CASTELLANI'S dysentery bacilli (*Bacillus parametadysentericus*), I can only repeat what I have already said, namely, that, to my mind, there is no satisfactory proof that it is in any way connected with dysentery, as we know it, at all. So far he has adduced none. It is not enough to affix a prefix of *para-*, *pseudo-*, or *meta-* to a hypothetical organism in order to connect it in any way with a disease. It is so easy to introduce organisms of this kind; so difficult to disprove them. I have always maintained that, were it possible to have a plate the size of a tennis court, and a platinum loop the size of a broom to spread the fæces, one would be able to isolate the true dysentery bacillus from every case. So far as I know none of these atypical organisms, so-called, have been isolated from the intestinal canal post-mortem.

Colonel CUMMINS has asked many questions, and I quite agree with him that in a war of immense proportions it is difficult to obtain a *reliable* opinion as to the most *prevalent form of infection* and act upon it *en gros*. A misdiagnosis in the first instance is not easily rectified. If I might tread on controversial ground, I might suggest that this is what happened during the early stages of the Gallipoli campaign, when every case of dysentery was labelled "amœbic," and the whole epidemic treated as if amœbic in origin—and I yet have to hear of a pure "epidemic" of this description. I should suggest that the best method of storage and distribution of anti-dysenteric serum would be to put it up into 20 c.c. phials and issue it to field ambulances, in either small ice boxes, or ones provided with a water jacket, which could be buried beneath the ground to keep cool. Such an equipment might take the place of the elaborate "cholera outfit" so admirable to look upon, but so seldom used. I do not think that, in the East at any rate, dust plays any part at all in the dissemination of dysentery; the sun's rays are great purifiers and kill the dysentery bacillus almost immediately. The reason for the severity of the bacillary dysentery in the East was because the majority of the cases were Shiga infections; I would therefore advocate the issue of a strong anti-Shiga serum to front-line units only, without the addition of anti-Flexner antigens. Sand, by causing irritation to the intestinal canal, also is a strong predisposing factor to the development of bacillary dysentery. I think the same irritation plays a part in the development of the amœbic disease, and explains why a proporti n of normal persons in a country like England may act as carriers of *E. histolytica* without developing any intestinal ulceration or hepatic abscess; in these "temperate" cases the protozoan acts merely as a saprophyte As to why the epidemic of dysentery developed so suddenly as it did in France is difficult to explain, save by the hypothesis that the Flexner-Y bacillus by frequent *passage* became suddenly exalted in virulence and that a large proportion of flies became naturally infective in consequence. There is also the question of susceptibility to be considered. Colonel CUMMINS found the Australians and New Zealanders much more susceptible to virulent infections than soldiers from England or other parts of the British Empire.

I am glad that Dr. WILLCOX appreciates the necessity of early treatment with serum and the advisability of giving sago and arrowroot as a routine diet in the disease. I might remind him that both these substances have had a considerable reputation as native remedies in the Pacific Islands and New Guinea, where they are at home. With him, I

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would like to see the consulting physician *browsing* more often in the laboratory and the *consulting* pathologist, when this genus is created in the next war, more often in the wards contemplating the source whence his material is derived.

As regards the existence of a pure malarial dysentery, I think this is a misnomer; when blood and mucus appear in the fæces of an acute abdominal malaria, it is due to actual hæmorrhages into the mucosa of the intestinal canal, but I do not consider the complication of bacillary dysentery and malaria a very frequent one. If the latter is of the subtertian variety, it is a very toxic and serious condition indeed. Whether the serum treatment aggravates a dysenteric polyarthritis is a very moot point indeed. I have written a paper on the subject which has lain dormant in the press for four years. I might say that arthritis occurs very frequently in some epidemics, quite independent of serum, and was known and described many years before the serum treatment was thought of.

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THE ETIOLOGY OF RHEUMATIC FEVER FROM A TROPICAL POINT OF VIEW. A FLEA THE PROBABLE CARRIER.

BY

J. TERTIUS CLARKE, M.R.C S., L.R.C.P. (PERAK).

In the search for the cause of a disease it is not uncommon to pay attention to the positive indications and to disregard the negative ; the absence, however, of a disease in a non-immune population may give a clue to its etiology. The cause of the disease may not exist, or, if the cause is a living organism, either the conditions of temperature and humidity may be unsuitable, or its proper carrier may be absent.

It is this reasoning which makes the study of the geographical distribution of disease of value, and has made me hope that the non-occurrence of rheumatic fever in the Malay Peninsula—and I believe in all the tropics at or about sea-level—might lead to the discovery of the cause of that disease.

Rheumatic fever has many of the characteristics of a specific disease. The manner of its onset and progress, its epidemic prevalence, the variation of intensity in epidemics, its seasonal waves, its more common occurrence in towns, its incidence on the young, and, finally, the fact that it does not occur in the Malay Peninsula, all point to its being a specific disease dependent on an organism of which the extra-corporeal existence can be carried on only under certain limited conditions.

In an article in the *Lancet*, of June 5th, 1915, entitled, "Rheumatic Fever and Rheumatoid Arthritis ; the Geographical Factor," an attempt was made to shew that these diseases do not occur in the tropics at or about sea-level. I gave in that article the experience of myself and of other medical men, and after quoting figures from the medical returns of several tropical countries, and those of the Malay States in particular, I shewed that the number of cases diagnosed in those countries as suffering from those diseases, or their sequelæ, was either nil or extremely small in comparison with the number occurring in Europe.

1. I then contended :—"In view of the number of cases in which the figure is zero, in view of my own experience and that of the writers I have quoted, and in view also of the small numbers diagnosed as valvular disease of the heart, the probability that the few cases given are either an error in diagnosis or in the subsequent transcription of the returns is greater than the probability that they are true cases of the disease."

2. There are many pitfalls in the way of the diagnosis of rheumatic fever, and the occasional description of a case, unless the whole syndrome is complete, should not be taken to disprove the statement that the disease does not occur in any particular locality. Any form of septic or toxic arthritis can easily be mistaken for acute rheumatism. A swollen joint, accompanied by fever, may be difficult to diagnose ; the anæmia of ankylostomiasis, with the fever of malaria, may be accompanied by swollen joints and cardiac murmurs ; dysentery is not infrequently followed by swollen joints and cardiac murmurs.

Rheumatic forms of typhoid fever are described[1] and a case of cerebro-spinal fever

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"simulating acute rheumatism" has been published[2]. CASTELLANI mentions that the condition in certain cases developing after a typical attack of dengue "may be indistinguishable clinically from polyarthritis of rheumatic origin,"[3] and cases of subacute osteitis may be accompanied by a cardiac murmur and have been mistaken for rheumatic fever.

3. It would be easy to add to the figures, but they can be found in the supplements to the *Journal of Tropical Medicine*. Nearly all the returns from the tropical colonies are similar; out of the thousands of cases treated in the hospitals, there are usually no cases of rheumatic fever, and very few of valvular disease other than atheroma, but the only additions I will make to the figures given in the article above referred to are the following:—In the State of Perak, during the years 1912-1917 inclusive, there have been 97,914 deaths recorded in a mean population of 548,606, and not one of these deaths has been ascribed to rheumatic fever.

I may add that Dr. J. T. DUNCAN, the Principal of the Medical School, Singapore, has kindly given me permission to state that he has not seen a case of rheumatic fever in Singapore, though he has been in charge of large hospitals, and that in about 600 post-mortem examinations he has not seen a single case of mitral stenosis.

The statement in Sir W. S. CHURCH'S article in ALLBUTT and ROLLESTON that "High and dry lands, when the temperature varies between wide limits in the twenty-four hours, appear to be particularly conducive to rheumatic fever," lends further support to the opinion that the disease does not occur in the tropics at or about sea-level. A disease which is most frequent in England, when summer has passed, is not likely to be common in those parts of the tropics where a heat much greater than the mean of English summers never ceases, and the dryness of an English summer never exists.

It must be mentioned that Drs. J. T. CALVERT and W. D. SUTHERLAND write:—"Although scarlet fever does not occur and rheumatic fever is not prevalent" (in Calcutta which is in the tropics) . . . . still we have met with young Bengali children suffering from rheumatic chorea with endocarditis (mitral), with rheumatic fever with fibrous nodules and endocarditis, and with rheumatic fever with well marked pericarditis and effusion"[4]. But Calcutta is on the edge of the tropics; not far northwards there is a large non-tropical and even cold area from which infection might be imported and the disease must be less common than is expressed by the words "not very prevalent," for, as shewn in my previous paper, out of 281 post-mortems at the Calcutta morgue, there was not one single case of endocarditis or stenosis of either the mitral or the aortic valve.

In England, it must be remembered that diseases of the circulatory apparatus cause probably 10 per cent. of the total deaths.

As scarlet fever has some bearing on my theory, it must be pointed out here that it is generally accepted that scarlet fever does not occur in the tropics except as an imported infection, and that it never spreads. Sir JAMES CANTLIE, in the year 1899, in the *Journal of Tropical Medicine*, was, I believe, the first to publish this observation. The absence of this disease from the tropics is mentioned by FOORD CAIGER in his article on scarlet fever in ALLBUTT and ROLLESTON, and by McCALLUM in OSLER and McCRAE.

Though this statement has been contradicted, and a few cases have been published, the fact holds with scarlet fever as with rheumatic fever, that should a few doubtful cases be shewn to have occurred, or even a few definite cases be proved, the statement that these diseases do not occur would for all practical purposes still be true.

4. The reason for the difference in geographical distribution of these diseases would appear to lie in the differences of temperature or of atmospheric humidity, excess or deficiency of either of which might be inimical to the existence of the causative organisms or to their carriers.

5. It is difficult to believe that the climate of the Federated Malay States is inimical to any kind of pathogenic micro-organism, but it is known that it is inimical to the life of the flea, and I suggest that the flea is the carrier of the organism which causes the disease rheumatic fever. Possibly the common human flea, *Pulex irritans*, which occasionally is found on rats, is implicated, but the common rat flea, *Ceratophyllus fasciatus*, which also bites man, may be more probable.

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The following are the facts from which this proposition is deduced:—

(*a*) Rheumatic fever is a disease of towns, and fleas are more prevalent in towns.

(*b*) The wave crests of scarlet fever and of rheumatic fever agree as to the time of the year in which they occur.

(*c*) In England a correspondence between the wave crests of scarlet fever and the highest prevalence of fleas has been shewn.

(*d*) Both scarlet fever and rheumatic fever are diseases of dry years.

(*e*) Fleas are more prevalent in dry years, and the highest incidence of both scarlet fever and rheumatic fever is in the period immediately following the dry months, *i.e.*, just after the highest flea incidence.

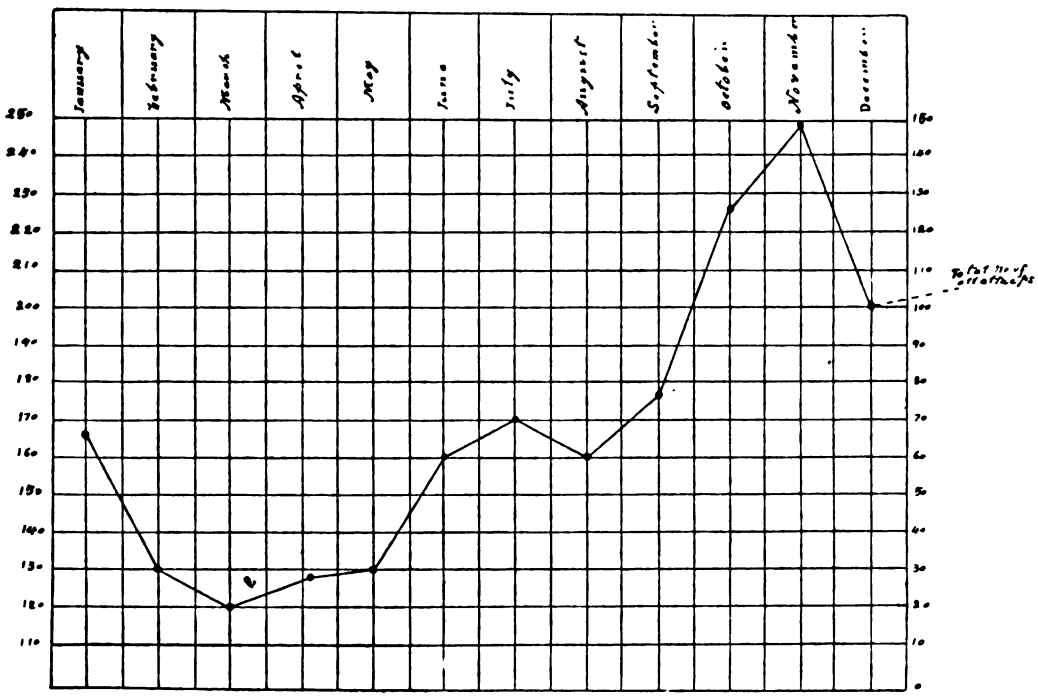
(*f*) Neither rheumatic fever nor scarlet fever occurs in the tropics.

(*g*) The number of fleas in the tropics is small compared with the numbers in Europe. The only flea which is at all common in the Federated Malay States is the cat flea (*Ctenocephalus felis*). The human flea (*P. irritans*) and the European rat flea (*C. fasciatus*) have not been found in the Federated Malay States (FLETCHER). *C. fasciatus* has not been found in India south of the United Provinces except in the Nilgiri Hills, and is never common in the United Provinces. In the United Provinces it is only found in the cold weather.[5]

(*h*) The highest number of fleas occurs in the period immediately preceding the greatest rise in the number of cases of rheumatic fever and scarlet fever.

To shew that the wave crests of scarlet fever and of rheumatic fever agree as to the time of year, I give two charts from ALLBUTT and ROLLESTON, which I am allowed to use by the kind permission of Messrs. Macmillan & Co.:—

CASES OF RHEUMATIC FEVER, LONDON HOSPITAL, 1873—1881.



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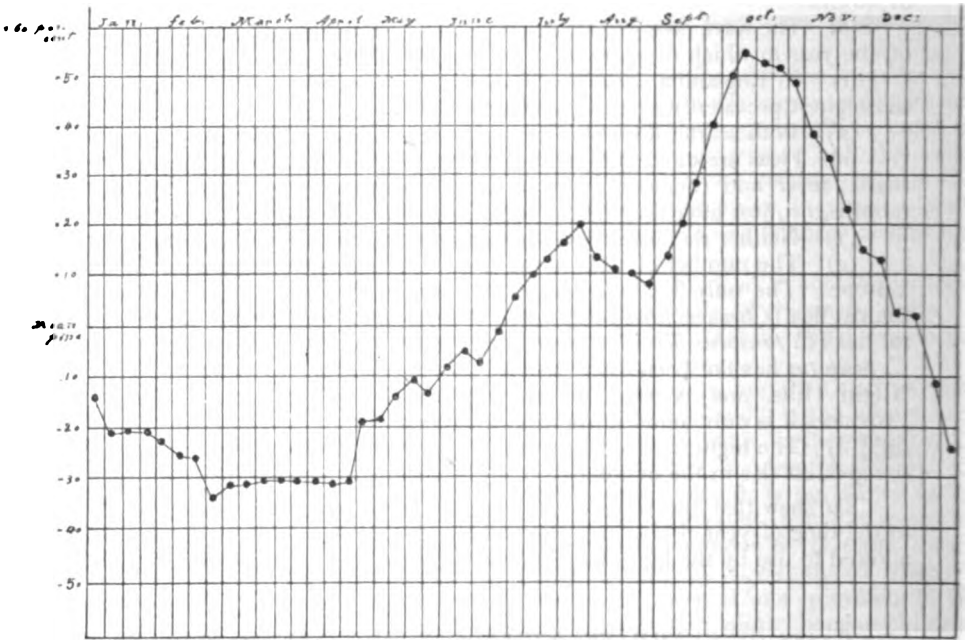
SCARLET FEVER NOTIFICATIONS, LONDON (15 years, 1890—1904).



The mean line represents an average weekly number of 368 notifications.

These charts cannot actually be superimposed, but have a remarkable similarity—a similarity so great as to suggest that the climatic conditions which lead to the one disease lead also to the other. Slightly altering a sentence in the article of Sir W. S. CHURCH'S, already quoted, it may be said that—

> The similarity of the seasonal variations of scarlet fever and rheumatic fever, and the similarity of their geographical distribution, supports the view that each of them is caused by a micro-organism capable of multiplication in a similar nidus outside our bodies.

6. For the correspondence between the wave crests of scarlet fever and the prevalence of fleas, I must quote from a leading article in the *British Medical Journal* of September 16th, 1916, on "Uncleanliness and Scarlet Fever," in which there occurs the following passage :—

> Now, in the current report of the Medical Officer of Health for London (Dr. HAMER) we have the results of a vermin enquiry ; they suggest that there may be a connection between vermin and scarlet fever. Two main preliminary conditions are mentioned. The first is that scarlet fever is wont to display major waves of prevalence, in which the intervals between the crests are some six to eight years. Such a wave crest was due in 1914, and appeared. A correspondence between this wave and one shewing the prevalence of fleas is found to exist. The second is that scarlet fever displays a remarkable inverse correlation with the rainfall. On the whole there is a general correspondence between the annual prevalence of dryness and scarlet fever. A similar correspondence has been noted in India for rat-fleas. In London it has been observed that the wave of flea incidence precedes that of scarlet fever by some three to eight weeks. 1914 was a hot, dry year.

7. Leaving out the remarks about fleas, and substituting for "scarlet fever" the words "rheumatic fever," these statements are almost identical with those of Sir A.

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NEWSHOLME, in his Milroy Lectures of 1895, on the "Natural History and Affinities of Rheumatic Fever."

8. Then if in England there be a correspondence between the wave crests of scarlet fever and of rheumatic fever, and a correspondence between the incidence of scarlet fever and the presence of fleas, there must exist the same correspondence between the incidence of rheumatic fever and the prevalence of fleas.

Sir A. NEWSHOLME,[6] in his Milroy Lectures in 1895, shewed that in epidemic years the mean annual temperature was as high or higher than the mean of a series of years; that epidemics of rheumatic fever occurred only when the mean earth temperature was over 50°F., and that the greatest epidemics occurred when the mean temperature had been thus high for two or more years in succession. He brought forward a considerable amount of evidence to shew that a low ground water level was conducive to, and a high ground water level inimical to, the occurrence of rheumatic fever. He concluded that rheumatic fever is drowned out in wet years and multiplies in dry years. These cold, wet years are inimical to the flea, while the conditions which NEWSHOLME describes as favourable to the occurrence of rheumatic fever are conducive to the propagation and survival of fleas. A relatively high—but not too high—mean temperature and a low level of ground water are conditions best suited to the flea.

Investigations have shewn the conditions which result in an increase in the number of fleas in the hot, dry summers of England.

It must be understood that the egg, larval and cocoon stages of the flea are not spent on the host, and that the insect stage is not passed entirely on its host, which it visits for feeding purposes. The imago of *X. cheopis* lives upon its host rather than in its nest, but this is not so with *C. fasciatus* or *P. irritans*.

The various stages of the flea are not all equally susceptible to risk, and the larval stage is the most delicate. All the stages are hindered by cold; "lagging" of the cocoon stage is easily induced; and it may be said that for *P. irritans* and *C. fasciatus*, there is an optimum temperature of between 55° F. and 75° F., with a humidity of 70 per cent., which is the most favourable, and that above or below these temperatures, and especially if the humidity is low, the conditions are unfavourable. Further, it may be said that the active larvæ can only withstand a narrow range of conditions and that a wet skin is fatal to this stage.

In the warm dry summers there would be less lagging of the cocoon stage; those which had hibernated for many months might develop, making for greater numbers. Warmth is favourable to the hatching of the eggs; a temperature of 40° F. will prevent the hatching of more than a small percentage, even of *C. fasciatus*. Only a few *P. irritans* will hatch at 46° F., while the eggs of *X. cheopis* will apparently not hatch below 55° F.

The rat is normally an inhabitant of the ground level or below it, and during the wet years the subsoil water is high. The larvæ which are hatched in wet years are in great danger of getting wetted, and so killed; whereas, in the dry years, the larvæ would have a better chance of keeping their skins dry and large numbers would come to maturity. In fact the fate of the larvæ is very similar to the fate of rheumatic fever, A low level of subsoil water is favourable to the life of the rat flea, *C. fasciatus*, as well as to rheumatic fever.

It is probable that this difference of ground water level is of greater importance than the difference of temperature; the mean of the 4-ft. thermometer of the hot years is only a few degrees higher than the mean of the average years; and temperatures at or about 50° F. are not fatal to any of the stages of the life of the flea, whereas wetting is immediately fatal to the larvæ, and the cocoon cannot withstand prolonged immersion.

STRICKLAND says, "there was a rise in the average number of *C. fasciatus* per rat during September,"[7] that is just before the occurrence of the greatest number of cases of rheumatic fever and scarlet fever.

In the Malay Peninsula the humidity is 80 per cent., which is not necessarily unfavourable to the flea during any of its stages, and the temperature, which varies between 69° F. and about 91° or 92° F., and occasionally two or three degrees higher,

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cannot itself, according to the observations of Mr. A. W. BACOT, of the Lister Institute, be said to be lethal to this insect.*

The mean temperature of the Federated Malay States is 80° to 81° F., and though it has been shewn that, if the saturation deficiency of the atmosphere is high, this temperature is detrimental to *X. cheopis*, and that *C. fasciatus* disappears, the same has not been shewn if the atmosphere is nearly saturated with moisture, as is the case in the Federated Malay States.

BACOT has shewn that *X. cheopis* has an optimum temperature of 15 to 20 degrees higher than *C. fasciatus*, and 5 degrees higher than that of *P. irritans*; and it may be that the mean temperature of the Federated Malay States, which during no month of the year falls below 79° F., may be higher than is compatible with the persistence of *P. irritans* and *C. fasciatus*, by reason of its never intermitting. The saturation deficiency in the Federated Malay States never rises to ·30 of an inch.

Anyhow, the fact remains that *P. irritans* and *C. fasciatus* have not been found in the Malay Peninsula, and the incidence of *X. cheopis* is only 1·042 per rat.

In the plague districts of Java the mean temperature is about 76° F., and the flea index on rats is a little higher than that of the Federated Malay States, and plague persists as it does in Mauritius, where the mean temperature falls to about 70° F. in the mean months of the year, and, as it does in Rangoon, where, though the mean is 80° F. for nine months of the year, during three months it is as low as 76° F.

It seems probable that a mean temperature of 80° F., with occasional rises to 94° or 95° F., is incompatible with the persistence of *P. irritans* and *C. fasciatus*, and is very detrimental to *X. cheopis*, whatever the humidity may be.

In the United Provinces of India the incidence of fleas on rats falls from about 10 in the cool months to about 2·5 in the hot dry months. The cool wet months in the United Provinces have about the temperature and humidity of the hot, dry summers of England. In the hot weather in the United Provinces the *C. fasciatus* is not found, nor is it found anywhere in India south of the United Provinces, except in the Nilgiri hills.

During a visit to the Rothschild museum at Tring, Dr. JORDON was able to shew me only two specimens of *P. irritans* from the tropics: one from New Guinea, which was found at a great elevation three days in from the coast, and one from Ghodasgaon in Khandesh, which, though at no great elevation, is only just inside the tropics, *i.e.*, about 20° north, and at an elevation of 800 feet.*

In fact, these two fleas may be said, as I have claimed for rheumatic fever, not to exist in the tropics at or about sea-level. And even should the presence of a few be demonstrated, small numbers would not affect my argument any more than would the description of a few cases of rheumatic fever.

It may be asked why I have selected the *C. fasciatus* as a probable carrier instead of other fleas. The reply is that it is the commonest man-biting flea, whose host is other than man, which is not found in the tropics. *P. irritans* is not excluded, but it seems to be probable if this disease is insect-carried, that there is another animal which is the habitual carrier. Both the dog and cat flea have been found in the tropics; the *Ctenopsylla musculi* is said not to bite man; and the fleas of those animals which are not found in houses need not be considered.

I have shewn that the conditions most conducive to rheumatic fever in England are the same as those most favourable to a high incidence of fleas; that these conditions do not and cannot occur in the Malay Peninsula, that *P. irritans* and *C. fasciatus* have not been found in that country, and that there is no rheumatic fever. Although I cannot claim to have proved it, I believe that these facts pertain to the whole of the tropics at or about sea-level.

It will be said that I have only shewn coincidences; but there is so strong a correspondence, both in time of the year and in place, of the occurrence of rheumatic fever and of fleas, and a similar correspondence in their absence, as to make it appear

* Ghodasgaon, or Ghorasgaon, is in East Khandesh, not far from Edalabad, in the Pūrna river valley. The altitude was left blank by the author, and has been estimated at the Map Department of the India Office, through the kind assistance of Mr. C. A. STOREY, Assistant Librarian.—*Ed.*

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that they occur in the relationship of cause and effect. Further, the rise and fall of each of them appears to correspond with the rise and fall of temperatures and humidities.

It is probable, as with plague, that the flea is only an intermediary, or carrier, and that the real host of the organism is an animal of which the flea is a parasite. That animal is most probably the rat.

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NOTE.

CONTRIBUTED BY DR. W. H. HAMER, MEDICAL OFFICER OF HEALTH AND SCHOOL MEDICAL OFFICER, COUNTY OF LONDON.

I gladly avail myself of the opportunity of writing a few lines on a flea hypothesis of rheumatic fever.

Dr. LONGSTAFF, in his original paper (*Epidem. Soc.*, 1880) on the relation between scarlet fever, puerperal fever and certain other diseases (one of which, it will be remembered, was rheumatism of the heart), commented upon the close resemblance between the curves of erysipelas and puerperal fever, and expressed the belief that they were "due to one poison"; he also referred to the inverse relationship exhibited in his group of diseases to rainfall. In later papers the facts have been brought, from time to time, up to date by Dr. LONGSTAFF, and the demonstration of the close relationship of the annual curves of mortality from scarlet fever, rheumatism of the heart, and the other diseases of the "scarlatinal group" to one another and to the inverted rainfall curve, is, of course, now generally admitted. As Mr. CLARKE points out, the similarity of the seasonal curves of scarlet fever and rheumatic fever has also been accepted since the date of Sir WILLIAM CHURCH's article in ALLBUTT and ROLLESTON's *System.* Mr. CLARKE gives a reference to my study of the relationship between the scarlet fever seasonal curve and the human flea curve, as observed in London. The data he alludes to have been collected during the past eleven years; the similarity referred to is very close, and the important fact emerges that the maximum for fleas antedates in each of the eleven years the maximum for scarlet fever, the interval between the two maxima varying from one to eight or more weeks. The curves have been published year by year (*Annual Report Med. Off. County of London*), and a summary of ten lines of reasoning which tend to support the conclusion that infected fleas produce scarlet fever, is given in the report for 1917 (p. 37), and the subject is further considered (pp. 44-49) in the report for 1918. Points of criticism presented by certain "school" and "return case phenomena," and by the relation of scarlet fever to social conditions, have engaged attention and, it is believed, have been met, but questions

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as to the correspondence in geographical distribution of scarlet fever and of *Pulex irritans* have been so far only cursorily considered. It is important that more detailed and precise knowlege of the distribution of scarlet fever and rheumatic fever and also of fleas should be obtained, and Mr. CLARKE'S paper will, it may be hoped, stimulate such enquiry. It must, perhaps, be assumed that in an age of devotion to laboratory methods, nothing short of ocular demonstration under the microscope or ultramicroscope of the actual organisms, or cultures thereof, will suffice to carry conviction ; but, in the meantime, it is worth remembering MATTHEWS DUNCAN'S saying, that experience shews there is nothing we are so likely to overlook as the things that are continually bobbing against our noses. Mr. CLARKE'S paper raises the question as to whether an hypothesis of flea-borne spread of rheumatic fever may not be one of those things.

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TREATMENT OF MALARIA, AND CHIEFLY OF CHRONIC MALARIA AND BLACKWATER FEVER.

BY DR. F. ROUX,

Formerly Médecin Inspecteur de l'Assistance Médicale Indigène à Madagascar.

If quinine constitutes the specific medicine for malaria, we must acknowledge that it is very often disadvantageous for the patients, either by itself or in consequence of the process of administration (ingestion, injection).

Besides, for patients suffering from chronic malaria, with hypertrophy of the spleen and the liver, quinine is almost always ineffective and very often dangerous.

The treatment I recommend serves the purpose of avoiding the disadvantages and the dangers of quinine. Before publishing it, I tested it during three years in Madagascar, Réunion and Algeria. It has been employed by many doctors at Haïti, Brazil and French Western Africa, and the results obtained have been satisfactory everywhere.

This treatment consists in intravenous injections of colloidal quinine, or *collobiase de quinine.*

Each phial contains—

Collobiase de quinine	2½ milligrammes.
Water	2 cubic centimetres.

For patients suffering from severe malaria, it is necessary to give every day or every second day, according to the gravity of the case, an injection with two phials, or 5 milligrammes of quinine.

For patients suffering from mild malaria one phial is enough.

In the majority of the cases it is needless to give more than three injections.

At what moment is it best to give the injection? For patients suffering from acute malaria, I advise, when it is possible, to inject the *collobiase de quinine* some time before the expected attack.

For patients suffering from chronic malaria one can give the injection at any time, but it is more advantageous to give it in the morning.

The patient must not breakfast before the injection.

The chronic patients are improved almost immediately. The appetite and the sleep come back very soon. The physician then thinks that the patient is cured, the more so as the hypertrophy of the liver and of the spleen decreases rapidly.

The cure is ascertained by the disappearance from the blood of the hematozoa.

The injection is generally followed, sometimes almost immediately, by a reaction which is very severe among some patients. This reaction is the stronger the more severely the patient is ill. It manifests itself by varied symptoms: headache, vomiting, lumbago, shivering, rise of temperature, sound sleep.

Among the thousands of patients who have been treated by my treatment we have not observed a serious symptom.

I insist upon this point: the treatment I recommend is chiefly efficacious with

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patients suffering from malaria for a long time. The older the cases the more efficacious is the treatment.

From my statistics no more than 3 per cent. of the patients have failed to be cured or very much improved by my treatment.

Treatment of Blackwater Fever.

Blackwater fever is observed only among patients suffering from malaria for some time. We had not till now a specific treatment for this fever. The doctors confined themselves to the treatment of the symptoms.

Therefore the rate of the mortality was very high, about 33 per cent.

I have tried at Madagascar a new treatment. It consists in intravenous injections of—

Colloidal arsenic (*collobiase d'arsenic*)	0·34 milligrammes.
Colloidal iron (*collobiase de fer*)	0·12 ,,
Water	2 grammes.

With Dr. Rabefaritra, I have treated 26 patients; 25 have been cured. The patient who died was a young girl, nine years old. I would not give the injection on account of the small size of the veins. I was obliged to substitute a hypodermic injection, which, I think, is useless

As soon as blackwater fever appears (and the main point is to give the injection as soon as possible after the appearance of the hæmoglobin in the urine), I inject into the veins a phial of the liquid above described.

Generally, twenty-four hours after the injection, the urine begins to become clearer. When I give a second injection, in the majority of the cases, the urine becomes clear some hours after.

The third day I give a third injection for precaution. After these three injections the hæmoglobin does not reappear, and the patient becomes convalescent.

Some days later, for about fifteen days, I prescribe 30 drops a day of adrenaline (solution $\frac{1}{1000}$).

During the convalescence, one generally observes an attack of malarial fever. In these cases I prescribe the injections above described for malaria.

They are without any inconvenience for the kidneys, and they cure the malaria.

I shall be very happy if the English doctors will try my treatment. I am at their disposal if they want any information or samples of the colloidal medicines I have used.

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Obituary.

SIR WILLIAM OSLER, Bart., M.D., F.R.S., F.R.C.P.

The passing of Sir William Osler is an event which affects the whole sphere of medical science. The specialism of to-day tends to isolate the keenest workers into somewhat narrow groups without that co-ordination and sense of proportion which is essential for the sound advance in the knowledge of human life, whether in health or disease, which is the proper domain of the practitioner of medicine.

Osler was a man who cannot be claimed as a distinctively tropical specialist any more than he could be assigned to any other of the great departments of medical science with which he was intimately associated. At the same time he may be singled out as the most conspicuous instance among leaders in the medical profession generally throughout the world, of one who realised the greatness of the discoveries of tropical medicine, associated himself with them and assigned them an adequate place in what may be called the system and practice of medicine. This is due to many causes: partly to his remarkable powers of judgment, his rigid impartiality, and his wide study of the whole range of his profession. But there were other reasons for this. It may be ascribed partly to his natural culture and varied educational opportunities, but it was also due to the fact that his main sphere of influence was in the New World, in a continent including the widest range of climate, and in which the great scourges of the tropics vitally affected every side of human life. Nor was this all; the period of his greatest contributions to science as a student, a research worker, a teacher, a writer, an administrator as well as a clinical physician, may be said to be between the years 1884–1904, during which the greatest triumphs of tropical medicine were unfolded.

An interesting evidence of what has been said above may be found in the arrangement of the first two sections of his *Principles and Practice of Medicine*, in the sixth edition, published in 1905. In this, the first section opens with "Diseases due to Protozoa," and the second section includes the "Fevers and other Infectious Diseases of the Tropics."

Osler's personal attractions, unfailing sympathy, genuine courtesy and unique influence are shewn in the numerous tributes to his memory which have appeared in many quarters. The son of a missionary to Canada, influenced as a boy by contact with the Rev. W. A. Johnson, founder and Warden of the Trinity College School, Weston, Toronto, one of the three men to whom he dedicated his great text book, he was a Christian gentleman of the highest type, whose loss has left us all the poorer, but who has handed on to us a memory and a tradition which may bear fruit for years to come in the profession which honoured him and of which he was a noble example.

Sir William Osler contributed to the Transactions "The War and Typhoid Fever." Vol. VIII., No. 2. December, 1914.

C. F. H.

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LIST OF FELLOWS.

The following were duly elected Fellows of the Society:—

Lieut. Colonel J. Allan Anderson, M.B., Ch.B., R.A.M.C.
W. Broughton-Alcock, London.
Walter Dawson, D.S.O., M.B., Lanark.
Susan A. Finch, M.B., M.R.C.S., Lucknow.
E. F. Hatton, M.D., late Colonial Surgeon, Grenada.
E. T. Jensen, M.B., M.R.C.S., Harley Street.
Lieut.-Colonel Clayton Lane, M.D., I.M.S. (retd.).
Donald Schokman, F.R.C.S.E., L.R.C.P. & S., Ceylon.
F. Roux, M.D., Paris.

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TRANSACTIONS

OF THE

SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

FEBRUARY 20TH, 1920.

VOLUME XIII. No. 6.

Proceedings of a Meeting of the Society held on Friday, February 20th, 1920, at 8.30 p.m., at 11, Chandos Street, Cavendish Square, W 1., Professor W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P. (*President*), in the Chair.

SMALL-POX AND ITS PREVENTION.

BY

COLONEL W. G. KING, C.I.E., I.M.S. (retired).

Discussion of subjects connected with Tropical Medicine and Hygiene being the rôle of this Society, Fellows may hesitate in accepting with equanimity the infliction upon them of a paper dealing with a disease recognised as present in the tropics, but which cannot be said to depend for its existence upon the special conditions there found. Without distinct proof, it is usual for writers to suggest that small-pox had its birth in the tropics; thus HIRSCH states, "The native foci of small-pox may be looked for in India and the countries of Central Africa." Be this as it may, there can be no doubt that for centuries it has been a disease of both temperate and tropical climates. In treating of this pest, therefore, facts throwing light upon its etiology may be of utility, whether gathered in Nyasaland or the South Coast of England. Indeed, whilst it is happily true that JENNER's immortal discovery has robbed small-pox of its terrors as a major scourge of mankind, its disfiguring, maiming and death-dealing power is still felt throughout the world.

The organisation of the United States Public Health Service permits of the movement of epidemic diseases being recorded with an accuracy which is not yet approached by other countries. In the Annual Report for that Service for 1916 (p. 110) this terse and decisive statement occurs:—"Small-pox is practically pandemic, its occurrence in greater or less degree being reported from all countries." Yet a recent work (*Half a Century of Small-pox and Vaccination*) by that able expert on the subject, Dr. MCVAIL, brings into prominence the fact that at least one member of the profession dealing with health matters in Great Britain, Dr. MILLARD, is satisfied that "small-pox, following the

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precedent of other diseases, is dying out, so far as this country is concerned" Consequently, he holds that the "danger of an infant contracting small-pox is no longer real. It has become so remote (and this is not because of infantile vaccination but because small-pox is leaving the country) that it is now a very debatable point whether the risk the infant runs from vaccination is not definitely greater than the risk from small-pox Moreover, one of the things we have learnt about small-pox is that where modern methods of prevention are carried out, it is essentially a disease of adults rather than children. It is spread in the factory, inn, lodging-house, etc., rather than in the home or school."

On the one hand, then, there is made the statement that small-pox is pandemic, and, on the other, that in that minute part of the world known as Great Britain—more especially that part of it known as Leicester—the disease is dying out; and so convinced is the authority concerned on this subject that he would no longer encourage infantile vaccination by legal measures.

In the present day, medical literature is the common property of the East and West, and it is therefore not surprising to find that Dr. MILLARD's contentions have been used by the exponents of anti-vaccination in the tropics, in supporting opposition to official efforts to spread vaccination. It is desirable, therefore, it be ascertained what are the conditions which have led to so favourable impressions and, if they exist, how far they are applicable to the tropics.

In the hands of anti-vaccinists, Dr. MILLARD's contentions are liable to be regarded as whole-hearted objections to vaccination. He, however, has made it clear that he is not a disbeliever in the protective value of vaccination in the individual, but argues that, in the presence of what is known as the "Leicester method," it is no longer necessary to urge infantile vaccination by legislative measures. Dr. MCVAIL, on the other hand (certainly in accord with the bulk of the medical profession), would regard the neglect of infantile vaccination as dangerous, in view of the impossibility of forecasting the future incidence of small-pox and its type when incident. Nevertheless, there is an agreement as to the validity of certain of the data concerned by both parties. In his review of the circumstances of Great Britain as to small-pox prevalence, Dr. MCVAIL arrives at the conclusion that "there has occurred during the last half-century a great diminution (1) in the fatality and (2) in the infectivity and the prevalence of small-pox." In illustration he affords figures which, in the hands of Dr. MILLARD, are compiled as follows:—"In successive decades, the deaths have been—1867-70, 58,614; 1877-86, 18,026; 1887-1906, 16."

Now whilst avoiding details in this controversy, it is well to hold in mind that Dr. MCVAIL, replying upon the splendid specimen of statistical labour by Dr. BRUCE LOW—*The Incidence of Small-pox throughout the World in recent years*—points to the existence of types of small-pox, and would assign the small mortality evident since 1892-93 in this country not to the "dying out" but to the advent of two different types of the disease. He shews the probabilities are that in the period 1902-05, the dominant factor in London was the "North African type," which, in 9,659 cases, yielded a fatality of 16·8 per cent, whereas, in the provinces, where the American type prevailed, in 23,883 cases, the rate was only 6·9 per cent.

As far as I can judge, Dr. MCVAIL confines the question of "infectivity" to the fact that in the "mild type" the *materies morbi* afforded by the scanty eruptions of the skin are less available for distribution than would be the case where the more extensive eruptions of the severe type are concerned. In this light, "infectivity" and prevalence have a necessary relation, without, I think, implying that the disease-bearing agent need in either case differ in capability of infecting the human being, should implantation be equally facilitated. If this be granted, should it be shewn that the "Leicester method" offers a mode of inhibiting prevalence superior to that employed elsewhere, Dr. MILLARD's contention would gain strength. There is, however, no reason to believe, in the present day, that in any country possessed of a technically trained sanitary staff similar sanitary methods, although devoid of the monopolising cognomen, are not in force. The difference in practice simply is that Leicester neglects to enforce by legal measures

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infantile vaccination, and trusts to the care of its sanitary staff for segregation of such cases as may occur. In the presence of the disease, however, vaccination of contacts is encouraged.

So long as sanitary staffs have, in common with their fellow mortals, the human failing of possessing various interpretations of conscientious duty and discipline, it must be held that to delay protection of the individual by vaccination till he becomes a "contact" (within a period of incubation open to chance) is to incur risk to human life decidedly greater than Dr. MILLARD regards as debatable in the practice of infantile vaccination. In the absence of the defensive line represented by a vaccinated population which encircles the little city of Leicester, Calcutta affords a demonstration of the inapplicability of the method in the tropics. In that city, in the five years preceding 1914, in 1,775 deaths of children under five years of age from small-pox, it was found that 80 per cent. were unvaccinated, whilst the risk in that city that Dr. MILLARD holds is such as to permit the death-rate between vaccinated (20 per cent.) and unvaccinated (80 per cent.) infants to be faced with equanimity by a Public Health Officer, is represented by two deaths in 224,471 cases of vaccination. One of these deaths was due to tetanus from which none of the persons vaccinated with the same vaccine suffered (the infinite probability being therefore that the disease was gained in the child's environment), whilst in the case of the other, which occurred two or three weeks subsequent to the operation, its connection therewith could not be fully proved. Nor is there any proof that, in accepting these elements of chance, there have been secured in Leicester results which would merit imitation in the tropics: notwithstanding that the experiment to which the "Leicester method" has been subjected, up to date, has been under favourable circumstances as to type of disease encountered. The pride of place cannot be given, as Dr. MILLARD would contend, to towns where the incidence of the disease (attacks) amounted to one per mille of the population, but to those localities where the disease having been introduced, was the more promptly extirpated. In this light, of fifteen towns of equal and greater population where small-pox made its appearance in the epidemic of 1902-05, Leicester takes the last place in order of merit as to prevention of spread; in twenty towns of less population, it occupies the eighteenth place No laboured comparison of the age composition of the respective populations is requisite, firstly, on account of their overwhelming numbers being fully representative of classes and occupations of the country, and, secondly, because Dr. MILLARD's main principle is that infantile vaccination, in the presence of modern conditions, does not influence results.

Of the inefficacy of segregation without vaccination, by a bill of £15,000 for temporary hospitals, the trammelling of trade by 2,000 attacks, and the evolution after this experience of not only trust in primary vaccination but a plea for compulsory revaccination by the Grand Jury, the Gloucester epidemic of 1896 furnished ample proofs. In Burma, according to COPE's *New History of the East Indies* published prior to 1781, segregation, necessarily then practised without the aid of vaccination, was no more successful than at Gloucester. In describing the country in the neighbourhood of Rangoon, he states:—"The country is fruitful and healthful . . . . but the small-pox is dreaded as pestiferous, and in the province of Kirian that distemper is most dangerous and most infectious, so that if any one is seized by that disease all the neighbourhood removes two or three miles distance and builds new houses. which is easily done with bamboos and reeds, which they have in great plenty. They leave with the diseased person a jar of water, a basket of raw rice and some earthen pots to boil it in, then they bid him farewell for twenty-one days. If the patient has strength enough to rise and boil rice, he may then recover, if not he must die alone. And it is observable that while a person has that distemper the tiger, for all his voraciousness, will not touch him."

The point of common professional interest in this controversy, however, is not the "Leicester method," but whether small-pox is a disease which, in the presence, and solely in consequence, of modern sanitary conditions, is "dying out"—a result which would not necessarily be confined to Great Britain where, up-to-date, there is no reason to regard the hygienic arrangements as unexceptionable. This is no new theory as to small-pox; and, in the early 'seventies, I was taught that plague also had been rendered impossible of

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prevalence. That both may be conquered is no idle dream of the sanitarian; but, pending an approach nearer to the millennium, when the sanitary requirements of both the individual and the community are fulfilled with a precision not yet attained, and disease spread from country to country by land, sea, subterranean or air traffic, is rendered impossible, it is likely to be dispelled by ever recurring harsh realities.

What evidence however is there available as to there being various types of small-pox and how far can the "dying out" theory be reconciled with their existence?

Types and Strains.

Until medical research shall have placed the profession more definitely than at present in a position to judge of the nature of the microbe or (as suggested by Koch) the protozoon which may be the causative agent of variola, epidemiological deductions aided by clinical observations (and also, I suggest, analogies derived from cultivation of variola-vaccine on animals) alone are available for guidance. *Consequently, virulence in individuals (case mortality or so-called fatality) seems to me the best available test of inherent qualities of type,* having regard to the fact that, although there must be a relation between facility of spread (infectivity) and virulence in respect to communities (incidence on populations) the factors determining the former are so numerous as to render it almost impossible to differentiate their value.

In such an inquiry one would naturally first ascertain what, if any, difference existed in the clinical character of the disease as described in periods long anterior to this, and, especially, as to its peculiarities immediately prior to the advent of vaccination.

Galen is credited with having dealt with small-pox, as he describes a plaster for the skin of patients apparently suffering therefrom. Mahomed Al Razi (known also as Rhazes), who died A.D. 932, describes discrete, confluent, and malignant varieties. In the last named, he represents the vesicular stage as exhibiting small hard and dry elevations, whilst the pustules assume a greenish, violet or black colour. He particularly urges care in treatment for prevention. He held that children are more susceptible to the disease than young men, and asserted that only exceptional persons once attacked in childhood suffer a recurrence.

The forms described by Rhazes do not differ in clinical characteristics from those mentioned by Surgeon Duncan Stewart, of the Honourable East India Company's Service, in 1844, when vaccination was making slow progress in India—subsequent to its introduction there in 1802.

One of the Medical Officers (Mr. O'Shaughnessy) who contributed to Duncan Stewart's Report, furnishes a striking description of the eruption in a hæmorrhagic case, which agrees with that afforded by Rhazes. He states that "the immature vesicles subsided and dried into rings of grey cuticle. The central points had sunk deep into the corium, appearing like a shower of ink stains upon a grey Turkey carpet." In a Calcutta hospital, Duncan Stewart found 32 per cent. were confluent cases. The hæmorrhagic form he terms petechial. Here the pustules are described as flat and imperfect many, however, being filled with blood; in these cases blood was present in the urine and stools, and exuded from the gums and fauces. In the malignant form "depressed spots were observed throughout the body similar to the small-pox." A case is described in which, in addition to hæmorrhagic exudations, the features were ulcerated, the back and hips sloughed, and the legs were flayed by vesiculations. In a post-mortem of a malignant case, within twelve hours of death, it is recorded that the pharynx and larynx were denuded of mucous membrane, "or, at least, this could not be distinguished from the pulpy offensive mass which lined its whole inner surface. From the *caput cæcum* to the sigmoid flexure the colon was studded with small conical ulcers standing out from a healthy base in distinct irregular eminences like small nipples—the apices crowned either with a pustule or with small round hollow ulcers holding a loose coagulum of blood." He adds that these appearances were quite distinct from the common ulcers of tropical dysentery or the phagædenic ulceration found in certain forms of tropical diarrhœa."

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DUNCAN STEWART asserts that those who have undergone the disease previously, naturally or communicated by inoculation, or who have been successfully vaccinated, if attacked suffer from the disease in a modified form. The after effects of small-pox are especially dilated upon; he found that cases of deep abcesses of the limbs, ulcerated cornea, putrid sore throat, and diarrhœa were common. Indeed, he cites a dispensary having a daily average of 280 patients, where not less than twelve would be found suffering from the "secondary effects of small-pox."

JURIN, who is usually quoted as an authority upon the subject as to English mortality, held that in pre-vaccination days the average fatality was between 16 and 18 per cent; but McVAIL has shewn that, amongst the cases considered there have been entered about 6,000 which had occurred in America, where a considerable number afforded a decidedly low rate. It is, I think, preferable where possible to trust to hospital statistics. The London Small-pox Hospital, which received naturally acquired cases of the disease, and, after 1746, cases of inoculation, exhibited in the 25-years' period ending 1800 a mortality of 25 per cent. Between 1836-51 separate records were maintained for unvaccinated cases; the mortality for this special class was found to be 35·55 per cent.; but, in 1863, the mortality was 47 per cent.

In 1844, DUNCAN STEWART, writing of the period 1827-44, as to the conditions of small-pox and vaccination in Bengal, quotes the case mortality in a Calcutta hospital as 32·3 per cent. These cases were presumably mixed instances of vaccinated, inoculated and unprotected cases as usual in the population at the period. In his Report as Health Officer, Calcutta, for the year 1894, SIMPSON tabulates 681 similarly mixed class of cases, and differentiates results in each; the mixed total representative of the population treated at that time afforded a case mortality of 38·8 per cent. In Madras City, between January, 1916, and August, 1917, in 1,491 cases the mortality was 32·1 per cent. In Rangoon, statistics extending from 1897 to 1916 exhibit, in a total of 15,456 cases, a mortality of 33·4 per cent. In both these cities the arrangements are such that cases are not likely to escape observation of the sanitary staffs, so that the figures have almost as good a value as hospital statistics. DUNCAN STEWART quotes the rate for 67 unprotected cases at 66 per cent. In 244 cases quoted by SIMPSON in the Report of 1894, just mentioned, the case mortality was 60·2 per cent. Dr. RAGHAVENDRA RAO, Health Officer, Madras City, in his Report for 1917, found in 511 cases of small-pox 21 per cent. unprotected; in these the death-rate was 65·74 per cent.

Evidently, therefore, in India, between the present period and 1884, no great change has occurred in the fatality rate, in at least the large areas specified. These rates contrast sharply with those quoted for Great Britain; but heavy as they are, there are countries within shorter travelling distances where grades of virulence are more markedly in evidence.

If the accompanying map be regarded, some idea of the comparative virulence of the disease in different parts of the world may be obtained, with probably less tediousness than were a recital of the bare figures trusted to. These are compiled from those afforded by Dr. BRUCE LOW's publication already quoted, and, in a few instances, from the United States Public Health Service Reports, and official reports issued in India and the Colonies. So as to maintain touch with Dr. McVAIL's statistics, they are *confined to the period of and subsequent to 1902.* Some countries evidently publish statements only of the total deaths, and give very scanty information or none concerning the fatality rate. Hence a uniform method has been pursued of finding an average standard figure as to fatality for a country from the largest and most reliable data available. In addition, or as the only mode of expressing facts, figures for special localities are mentioned: these are distinguished by spots. Where vaccination is compulsory that fact is intimated by a Geneva cross; where compulsion is well exercised this cross is enclosed in a circle; where vaccination is recognised officially but not enforced by law, an open black circle is used; where enforced in special areas only, the circle is enclosed within another circle.

In attempting to draw conclusions from the teaching of this map, the more obvious points which require consideration would be (1) possible modes of transfer of

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the disease from country to country; (2) the influence of (*a*) climate on the *materies morbi*; grades of resistance to small-pox virus (*b*) inherent to individuals and races and to communities whose well-being is protected (*c*) by sanitary care of their environment, and (*d*) by artificially increasing resistance of its members by anti-small-pox vaccination.

(1) The first item need not be followed in detail—its possibilities are obvious in the transit of travellers during incubation by trade and pilgrim land routes, and the carriage in their personal effects and in merchandise of the *materies morbi*; as to sea routes, the map exhibits distances between ports throughout the world which should bring to mind how readily—irrespective of future ærial traffic—within a twelve-days' period, infection might be similarly transferred from country to country by shipping.

(2) As to (*a*), there is nothing on record to shew that small-pox need succumb to the combined conditions known as "climate" of any particular place; although facility of spread is influenced by temperature and moisture. DUNCAN STEWART well and concisely defines the conditions; he states, "the violence of the epidemic is moderated by a damp hot atmosphere, its diffusion promoted by dry and cold weather." The subjection of raw unprotected vaccine to heat and moisture is inimical to its vitality. In days prior to the use of preserved vaccine, officers working in the hot and damp climates of Moulmein, Rangoon, and the Malabar Coast, declared the task of prosecuting vaccination duties was thereby much trammelled. If inoculated calves be retained in a high temperature, and an attempt be made to secure coolness by injudicious overloading of the air with moisture (as in the use of sprays, "tatties," and the like) degeneration of the vaccine occurs. The retention of dryness with coolness favours the vitality of desiccated vaccine, and was the ideal aimed at when scabs for inoculation or for vaccination were employed. Rangoon is within the wet, Mandalay within the dry zone of Burma; the average fatality rate for the unvaccinated in the dry zone is markedly the greater.*

(*b*) Neither in man nor in the animal does the same vaccine produce precisely similar results. Individual peculiarities thus exhibited are usually regarded as due to varying grades of resistance to the virus. The dark races have long been credited with special susceptibility. If ill-conditioned animals be employed from generation to generation—especially if in a high temperature they be retained in an environment in which the skin is liable to defilement—there result hastened maturation of the vesicles and inflammatory and purulent conditions of the skin.

In small-pox, not only are differences in action of the virus evident in respect to individuals, but also in communities; so fully is this the case that it has become necessary to refer to these differences as incident to types and strains. In using these terms, either in respect to vaccine or to small-pox, I would restrict the term "type" solely to the characteristics of the original source - the parent stock; and "strains" to the descendants of the parent stock, as modified in characteristics by conditions incidental to their transmission from subject to subject. The stock type of vaccine, and possibly of small-pox, never fails to exhibit more or less its original character in its strains from generation to generation. Indeed, if the type be doomed to die out, it would, I believe, still exhibit in a weakly strain some peculiarity of the old stock. For example, Assistant-Surgeon ESKEY, U.S.A., has shewn that in certain families consanguineous qualities may predispose to the hæmorrhagic type of small-pox. A weakly strain of small-pox might well on such soil regain its old virulence.

(*c*) Virulent types of small-pox exhibit much skin disturbance, although the virus is credited with having a greater affinity for the internal organs than vaccine, which latter shews a marked preference for pavement epithelium. NOGUCHI based his successful efforts for the production of purified vaccine on this fact, and used the testicle of the rabbit, with its limited epithelium, as favourable for cultivation purposes. With this vaccine implanted on the skin, there is a minimum of areola - the inference being that the greater the number of extraneous organisms the more the potential aggravation of

* See attached tables. The facts merit further enquiry. The age populations differ very greatly. At the same time the records of 1918 and 1919 shew marked differences in local virulence in Rangoon.

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skin action and purulent tendencies. Similarly, it would be reasonable to suppose that if, time after time, in transmission of small-pox in a community the *materies morbi* were in association with organisms specially incident to dirt, there would result increased skin action which would add to the destructive properties of the virus. An illustration of the possibility of associated organisms exhibiting their influence in one generation and, during transmission in another, failing to exhibit themselves, and yet in another, again making an unwelcome appearance was forced upon me by rather a hard experience.

At an early stage of my vaccine work, and before I had learnt the necessity of always reserving a supply of stock vaccine for emergencies, I used for the accommodation of a number of inoculated calves a building with a cement concrete floor which had not been fully seasoned. A flood then occurred, resulting in the inoculated surfaces being wetted with the general contents of the surroundings. On maturation the vesicles were found fairly developed, but there was a general inflammatory tumefaction. The vaccine was, of course, unfit for issue; but I determined to see whether I could not, by judicious selection of vesicles from generation to generation, get the strain back to stock type. The result was that a reversion to the type was duly secured; but, time after time, just as hopes were apparently about to be fulfilled, by a permanent absence of the unwonted inimical factor, one or more vesicles in the whole mass would exhibit the inflammatory condition. Although I believe that by means of selection the vaccine type could have been attained it was, for practical purposes, hopeless.

It was in dwellings of an extremely insanitary and overcrowded nature that DUNCAN STEWART found the very virulent forms of small-pox to which I have alluded, and he insists upon the favouring influences for malignancy found in insanitary conditions.

I would, therefore, suggest the hypothesis that the influence of sanitation upon strains and types of small-pox may make itself evident by limiting the number and, especially, the nature of associated micro-organisms likely to be transferred with the *materies morbi* of the skin of small-pox subjects to their fellow beings. Indeed, I think, instead of waiting to use tinct. iodi. and other antiseptics which have been suggested for application to small-pox vesicles, it would be advisable, between the papular and vesicular stage, to specially prepare the skin for the ordeal it has to undergo by purification—without subjecting the patient to chill—a possibility with some gaseous preparation.

If these considerations be applicable, the deduction may be made that if human beings in states of low vitality resulting, say, from ill-feeding, be placed in overcrowded and filthy dwellings, and amongst them a strain of small-pox be introduced, in the course of transmission, there will result an alteration of its characteristics in the direction of virulence if the strain at the time of introduction be mild; or it will simply retain its mild or virulent properties as introduced if the conditions to which it is submitted be those under which it has hitherto maintained its vitality.

That small-pox epidemics do vary in virulence has long been recognised. Both JENNER and SYDENHAM held that, at times, small-pox assumed a very mild character. Dr. BENJAMIN RUSH, a Professor of the University of Philadelphia, writing in 1794, states: "Small-pox, even in the natural way, does not always confine the patient." In another connection he states, "The weather in April resembled that of March in common years. . . . The small-pox by inoculation was more mortal than in former years." PRINZING gives a definite instance of increase of virulence during the course of an epidemic in 1870-71. "It was observed in Lyons, as in other places, that not only the number of persons who contracted small-pox, but also the virulence of the disease itself increased; whereas only 10·6 per cent. of 227 sporadic cases resulted fatally between January and September, out of 1,004 who contracted the disease between 1870 and July, 1871, no less than 21·7 per cent. died." The Reports of the Health Officer for Madras City for 1916, 1917 and 1918 shew a steady increase in virulence in an epidemic of small-pox during those years. As in most tropical cities, including the so-called "City of Palaces," the insanitary areas there are frequent, and form the favourite foci for small-pox. In 1916, in 1,489 attacks the deaths were 32·0 per cent.; in 1917, in 582 attacks the deaths were 33·5 per cent.; and in 1918, in 677 attacks the deaths were 40·2 per cent. These results were secured in a population which, as in the case quoted by

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PRINZING, was but partially protected by vaccination. It is probable also that its resistance was impaired, in common with populations throughout the world following war conditions, by decreased choice of articles of diet, as was the case with the French population of 1870-71, dealt with in PRINZING'S figures. A further instance of increase of virulence displayed in the course of an epidemic is quoted by BRUCE LOW. In 1910, Montevideo had an outbreak of small-pox, in which, in the first 108 cases, the fatality was 22 per cent., but by June, 1910, the rate had increased to 42 per cent. The probability was, in this case, that insanitary conditions in town-life had conduced to this result, as it is recorded that in 678 cases outside Montevideo the rate was only 17·4 per cent.

(*d*) Although, in the absence of revaccination, infantile vaccination has a value decreasing with the years after puberty, there is ample evidence that a beneficial modifying influence persists. For instance, Dr. BARRY'S Sheffield Report shews that in persons over ten years of age who had been vaccinated once in infancy, the attack rate per 100,000 of the population, resident within actually invaded houses, was 281 against 696 of the unvaccinated; whilst the death-rate of the former was only 14 against 371 of the latter. As already stated, DUNCAN STEWART, in his Report, particularly invites attention to the fact that, where the vaccinated were attacked, the clinical symptoms up to the stage of vesiculation seemed as threatening as in the unvaccinated, but that these quickly subsided; whereas, in the unvaccinated, there was no hope of other than slow progress to convalescence. The same observation was made by Dr. PRIESTLY, the Health Officer of Leicester, in his account of the 1892-93 epidemic in that city, given at the annual meeting of the British Medical Association of 1894. If, then, small-pox were transmitted in a community whose resistance had been, in whole or in part, raised by vaccination, not only might it be expected that the total attacks and fatality therefrom would be less than in communities where this condition had not been attained, but that the strain of small-pox, *in proportion to the continuity with which it encountered resistance during transmission in individuals*, would undergo attenuation in virulence.

I think the map shews that the various factors I have adverted to are in operation, in more or less degree, in the countries of the world.

Within the portion barred from right to left are the chief countries of Europe; the vaccinal status by conventional signs, and fatality of each, is shewn. Germany (10·8) is possessed of a population better vaccinated than that of certain other European countries, but still exhibits weakness in its vaccination of females as compared with males, and in giving too great license in the period for the accomplishment of infantile vaccination. It nevertheless makes an excellent defence against invasion by its immediate neighbours, Russia (38·8) and Austria, where, for some years before 1907 and the occurrence of an epidemic, antivaccinists had been busy. Professor SOBERNHEIM credits Austria with 100,000 attacks in 1915, with a fatality of 23·5. Sweden, the first country to adopt compulsory vaccination, had only eleven deaths between 1902 and 1916, and the fatality rate, therefore, is practically *nil*. Norway, in spite of much intercommunication at its ports with Russia, has a fatality rate of only 7·9. France (8·4) learnt from its 1870-71 experience that prevention is better than cure; and during the late war has demonstrated, both in its army and civil populations, infinitely better results than at that period. Belgium (16·3), where the public health authorities content themselves with merely recommending vaccination, and Luxembourg (19·7) afford marked contrasts to their neighbour Holland (8·4), which is "a well-vaccinated country" (BRUCE LOW). England and Wales exhibit a fatality in the provinces of 6·8 against 15·3 in London. Dr. MCVAIL, in his dealing with the "Leicester method," makes a special point of this difference, by suggesting the introduction of the American "mild type" of small-pox in the provinces and the North African type in London.

It is evident that, within the right to left barred area of Europe, the fatality rates are materially below those recognised as normal in Europe before the introduction of vaccination. In this area also, sanitary organisation receives more attention than elsewhere.

When, however, the extreme East is examined, it will be observed that the rate recorded by DUNCAN STEWART in India, in 1844, still prevails in the huge areas of China,

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India and Burma; and that using the fatality rate for the unvaccinated or otherwise unprotected as found in India—and regarding the rate as shewn for Hong Kong (76·4) as implying that deaths have been reported out of proportion to the actualities of attacks—it still remains over 60 per cent. Japan adopted compulsory vaccination in 1885. The first appreciable influence has been the limitation of attacks. A decline of virulence would now also seem approaching. From 1902 to 1915 the fatality rate was 31·3; in 1916-17, it was 22·8. In the great expanse of Asia there is one other exception to a general fatality rate of over 30 per cent., namely, Baghdad (24).

Baghdad city serves as a great central market for neighbouring countries—one at least of which, from the small-pox point of view, must be a source of danger. Dr. BRUCE LOW records that at the International Teheran Sanitary Council in 1907, it was stated that Persia loses from 50,000 to 100,000 annually from small-pox and that thousands lose their sight from the disease. My son, Dr. RALPH KING, who was with the forces in Mesopotamia, informs me that though the Turks had neglected infantile vaccination during the war, previous to that period it had been fairly well maintained—in marked contrast to other areas under the Turks; and it was locally understood that this special care was exercised having regard to the fact that Baghdad was a great caravan centre. It will be remembered also that it was *via* Baghdad, in 1802, that India received its vaccine, through the exertions of Dr. JAMES SHORT, then the Residency Surgeon. It is possible the influence of his pioneer work in Baghdad still survives. Nowhere else where the Turks or Arabs are in predominance is such a favourable rate found.

The conditions found in North Africa are of interest in connection with the so-called North African type, which is credited with the 15·3 fatality rate in London, in contrast with 6·8 of the "mild American type" which prevailed in the provinces. Dr. KERR, a missionary in Morocco, has stated that in Rabat, which had a population of 26,000, there occurred in 1894 over 1,000 deaths from the disease in two months (*British Medical Journal*, 1894, p. 1495). In Tunisia, in 1917, the fatality was 40·4. This contrasts strongly with the rate found in Algiers (12·7); but here the French authorities are alive to the necessity of pushing vaccination. The Institut Pasteur, under its energetic Director, Dr. EDMOND SERGENT, issues annually about 600,000 vaccine portions. The interest in the North African rate (assuming the Tunisian figures as representative) is that it forms a danger to France and through that country to England. In 1906-07, Marseilles was attacked with small-pox, which gave a fatality rate in 5,695 attacks of 41 per cent. The city contained a considerable number of unprotected persons, owing to an anti-vaccinist prejudice. There is reason to believe that the disease was imported from the North African area. In 1906, Paris had a fatality rate of 3·8, but in 1907 (synchronously with the Marseilles epidemic) this was raised to 10·2; between 1908 and 1913, in three years it did not reach 3 per cent., in two did not reach 7 per cent., but in 1910 attained 9·4 per cent. In 1900-01 (the Exhibition years) the fatality was 16·7 per cent., workmen from Marseilles being the starting point of infection. Again, in the 1901-02 epidemic in London, with its fatality of 16·8 per cent., Sir SHIRLEY MURPHY (quoted by Dr. MCVAIL) asserts that small-pox had been frequently introduced from Paris, and to this Dr. MCVAIL adds the comment that "Paris and Marseilles were the chief centres for spread of infection in France at this time." The matter to which I would, however, invite attention is that just as Baghdad (with frequent opportunities for infection from unvaccinated Persia, from Tripoli (40·3), Beirut (42·2 to 50·8), Beirut (39·2 to 42·2), Damascus (50·8), and Turkey (37·2), possesses a population sufficiently protected to attenuate the virulence of imported strains of small-pox of this character so fully as to be represented by a fatality of only 24 per cent., so Marseilles, at that time (1906-07) largely unvaccinated, on receiving infection from North Africa as typified by Tunisia (40·4) failed to secure any attenuation and, indeed, perpetuated the strain by yielding a fatality of 41·4 per cent. in 5,695 cases. Yet, when the same small pox strain was transferred to better vaccinated Paris, the fatality in 439 cases was only 10·8 per cent. Importation of the same strain in London in 1901-02 produced, in 9,659 cases, a fatality of 16·1 per cent. Although, as Dr. MCVAIL contends, there prevailed in England during 1902-05 the "mild type" of small-pox, having regard to the fact that the case mortality rate of the unvac-

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cinated in the provinces was 19·3 per cent.—which rate would be incompatible with that type – it is probable that the virulent North African strain introduced into London spread also to the provinces, and there exhibited further attenuation by transmission through a population which had been largely protected by infantile vaccination.

Along the East Coast of Africa, the fatality does not differ materially from that found further East; on the West Coast, the fatality corresponds to that found in the less protected parts of Europe.

The map shews the United States, Canada, British Guiana, New Zealand, certain of the West India Islands, a part of South Africa and of Australia barred diagonally from left to right. Within these areas, there has prevailed from time to time an affection which is regarded as a "mild type" of small-pox. A very careful and comprehensive investigation by Dr. ARMSTRONG and associated officers of the Public Health Service of New South Wales, confirms the opinion that the disease is a type of small-pox. They assert that "it is due to the same organism as small-pox, that it is probably due to a mutational change in this organism by which its virulence is diminished and possibly some phase of its life history eliminated, and that it is probable that it is unlikely to recover the general intense virulence of ordinary small-pox."

In the United States, in 386,341 cases, the fatality was 1·8 per cent. In New South Wales, in 1,073 cases, there was but one death. Dr. PATON, the Director-General of Public Health, in his report for 1917, states in respect to this disease (which first shewed itself in New South Wales in 1913). that "the type of the disease shewed no variation from the mild form in which it has shewn itself throughout the epidemic, and no deaths were recorded from it."

In Basutoland, Bechuanaland and Cape Colony also a markedly mild type of the disease has existence. The fatality rate in the unvaccinated in Cape Colony in 1903, amounted to only 4·3 per cent.; in a mixed population of vaccinated and unvaccinated, 3·8 per cent. In Basutoland in 1912-13, a fatality of 6·6 per cent. in 600 cases was attained. In 1916, in Bechuanaland 20,000 natives were attacked.

The origin of this mild type of small-pox has not been traced. Dr. ARMSTRONG, in his report, quotes VAN SWEITEN as referring in 1759 to an outbreak, "which was so mild that secondary fever is not manifested, and constantly is wanting, convalescence coming on the eighth day of the eruption," and adds that this type attracted notice in Canada and the United States about 1896. Dr. BENJAMIN RUSH, of Philadelphia, whom I have already quoted, evidently dealt with a very mild type of small-pox which by 1794 must have been fully recognised. He says: "The small-pox was once as fatal as the yellow fever and the plague. At present it yields as universally to a vegetable diet and evacuation in the hands of apothecaries, the clergy, and even good women, as it does in the hands of the Doctors of Physic." He was at heart a sound sanitarian, for he adds: "They have narrow conceptions not only of divine goodness, but of the gradual progress of human knowledge, who suppose that all pestilential diseases shall not, like the small-pox, sooner or later cease to be the scourge and terror of mankind." This prevalence of a mild type of small-pox might well lead to the opinion, where attention is confined to a single locality, that small-pox is "dying out" in the country concerned—as indeed, implied in regard to Leicester, when Dr. MCVAIL suggests, in refutation of Dr. MILLARD's theory, that the American mild type of small-pox was that prevalent in the provinces of England, in the epidemic of 1902-03. Curiously enough, Dr. MILLARD's objection to infantile vaccination is that it renders cases so mild that medical men cannot readily arrive at a diagnosis;* whereas, the New South Wales report referred to states that no difficulty, "with very few exceptions," was experienced in 3,000 instances in differentiating small-pox and varicella; and that the "unvaccinated" form the real danger, because unless the cases are sufficiently severe to attract attention the disease has the opportunity of spread at the outset.

But America has not only the mild type of small-pox to contend with. The various Independent States of the South of America have, except in the midst of epidemics, made

* Drs. NIVISON FORCE and H. L. BECKWITH have proposed an allergic test, which should be useful.

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little progress in vaccination, notwithstanding that Uruguay, in 1910, after going through an epidemic with a fatality of 42, made vaccination compulsory with calf lymph within the first six months of life, followed by revaccination at 10 and again at 20. Very little information is available as to America's near neighbour Mexico, but by compiling the few figures obtainable by Dr. BRUCE LOW I find that, in nine large towns contributing a total of 2,516 cases, the general fatality was 48·8 per cent. Invasions of America by this type of disease occur at not infrequent intervals, but have apparently been met by prompt vaccination campaigns which have stayed the progress. The U.S.A. Public Health Service Report of January 29, 1915, in reference to small-pox derived from this source states, "Most of the cases have been of the hæmorrhagic type with the exception of the varioloid cases, all have been of the hæmorrhagic or confluent types." Surgeon CREEL describes one of these virulent cases thus: "Until the day of his death no eruption had developed, but, on the morning of January 15th, ecchymoses and petechiae began to appear, and shortly afterwards he had severe hæmaturia and died at noon. The body presented no evidence of macular or papular eruption. It was very frankly a case of *purpura variolosa*." Further, there had been several illustrations of the fact that Australia is open to invasion by small-pox shipborne from European and Asiatic ports, all of which have been promptly dealt with at the ports concerned. In the Philippines, a former fatality of 40 per cent. is now, as a result of vaccination, about 9·8 per cent.

If, as I have attempted to claim, temperature and moisture incidental to climate, the inherent grades of resistance of races, the natural and acquired resistance of individuals (in the latter case due to vaccination, inoculation and previous attacks) and the several factors concerned in the health and the state of sanitary advance of communities affect the grade of virulence of small-pox, it suffices that after drawing your attention to the chief figures representing fatality exhibited on the map, I leave unsaid where such influences are likely to be predominant. Those addressed, from their knowledge of the circumstances affecting the eugenics of the areas concerned, may well conform to or disallow my deductions.

The total data available do not suffice to form final conclusions, but point to the existence of grades of virulence of small-pox recognisable as of two types, two strains and one substrain :—

(1) The most virulent form of small-pox is found east of long. 40° E. and between lat. 40° N. and 6° S. Amongst Asiatic populations, the general fatality rate per cent. (that is, as found in populations where the protected by vaccination, inoculation and previous small-pox are in various proportions) ranges from 33 to 50; in the vaccinated of all ages, from 30 to 60; and in the unvaccinated from 60 to 80. The disease in this form may be conveniently recognised as of the "Eastern type." Its characteristics are in accord with the earliest records.

(2) To the west of long. 40° E., during its gradual transference from Asiatic races and tropical conditions, the Eastern type loses much of its virulence, and forms a strain capable of (but very rarely) reverting to the virulence of its parent stock. It extends throughout parts of the world largely inhabited by Europeans and their descendants. It might be termed the "Western strain" of small-pox. The general fatality in pre-vaccination days was from 18 to 25; since this period, in the vaccinated, from 1 to 5, and rarely over 7 per cent.; in the unvaccinated, from 19·3 to 47 per cent. Its characteristics have not altered since the period of the earliest records.

(3) Within the area barred from right to left, as shewn on the map, are included countries in Europe where the general fatality rate is smaller than in any part of the world where either the Eastern type or its Western strain is evident. These, more or less, consistently carry out vaccination of their populations, with the exception of Belgium, where vaccination is merely "recommended" by the public health authorities; it, consequently, presents an object lesson when contrasted with its close and well-vaccinated neighbour Holland. In Belgium the general fatality rate is 16·3, in Holland 8·4 per cent. Within this barred area, the general fatality rate (and probably the fatality rate of the unvaccinated) presents sufficient contrast to the Western strain in other parts of the world to merit recognition of the existence of a "Western substrain" of small-pox. Within this

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barred area the virulent Eastern strain fails in the course of successive transmissions to maintain its pristine virulence.

(4) In South Africa there exists a second type of small-pox distinguishable both from the Eastern type and its Western strain by its mild constitutional symptoms, though attended with considerable skin disturbance. It here affords a general case fatality of from 3·8 to 4·3 per cent Probably from this source has been evolved a strain of less virulence, which has exhibited itself in races of European origin in America, Canada and Australia. In the former two countries the general fatality rate is about 1·8 per cent; in the latter, the rate is somewhat less than 1 per mille. The South African form of original virulence may be recognised as the "amaas" type and, as exhibited in the countries mentioned, the "amaas strain" of small-pox.

The Position of Vaccination.

The power of vaccination to protect the individual and to prevent spread in the community requires no emphasis; in the individual, the extent to which the system has been brought under the influence of vaccine—a matter having a probable relation to the strain of vaccine used, and the grade of reaction which has been ensured by a certain minimum number of successful insertions—the extent to which this prophylactic measure has been adopted by the mass of the community, and the average period which has elapsed since its adoption, are the facts involved. Of recent instances of the prevention of spread of epidemics by intensive vaccination campaigns, I may quote the work of Dr. Bousfield at Khartoum and its vicinity, in which 82 per cent. of the population were vaccinated or revaccinated; in Manilla, where the occurrence of sixteen cases of small-pox was followed in the Second Quarter of 1919, by 60,714 vaccinations; in Nyasaland, under Dr. Hearsey, where the rapid push of vaccination stayed an outbreak amongst followers proceeding to the East Africa war area; and, in Paris, under L. Camus, where, during the late war period, 1,374,000 vaccinations were performed, with the result that there were only 56 cases of small-pox, against 15,000 deaths from that disease in the 1870-71 period of war and of neglected vaccination.

To enter into the pros and cons of the question of age would unduly prolong this already lengthy paper, and I therefore avoid details. Much confusion has crept into the question by statements being made as to the number of persons dying at such an age, in relation to the question as to whether they have been vaccinated or not; but it is exceedingly rarely, so far as I have been able to ascertain, that these statistics have been made in terms of rates on the respective age periods of the populations concerned. It has been sought to shew that, at particular periods of life, the incidence of small-pox upon certain ages is less than at others. To ascertain the truth conveyed by such estimates, there would be requisite a very full history of the vaccination and small-pox incidence in a series of years amongst the people concerned. Further, such statistics never agree upon one evident factor not yet standardized, namely, the duration of the immunity afforded by vaccination. So far as my reading of statistics with which I had to deal officially goes, for all practical purposes, the vigorous age of life may afford both to the vaccinated and unvaccinated a better fight with death than at the very early or late periods of life, but, failing either previous small-pox, inoculation or vaccination, all ages are equally susceptible to attack. If this be so in tropical countries, where the presence of small-pox has not been brought very fully under check by vaccination and sanitary measures, it would be erroneous to advise that the ruling of the 1898 Vaccination Act of England, which prescribes compulsion at six months of age, be followed, and more so that a ruling (still in force both in Germany and Japan) that choice of period be permitted up to the completion of the first year of life, be adopted. The old Act of 1867, of England, rightly laid down that vaccination be performed within the first three months of life. In the Madras Presidency, vaccination in the rural areas is not compulsory; in municipal areas it is so. In the decade 1882-92, I found that (compulsion in all municipal areas not then being complete but still existing in many) death incidence from small-pox under one year of age was 2·7 and from the one to ten years' period, 0·69 per mille, against, in rural areas, 8·1 and 0·7 respectively.

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These figures shewed that whilst within the first year of life the waste of life in rural areas was vastly greater than within municipalities, both areas levelled up fairly well by the time the ten years period was completed; in the interval, vaccination under the persuasive efforts of vaccinators had added to the protection gained by naturally acquired small-pox in surviving children.

In 1913, in the presence of a much improved general grade of protection by vaccination than in the decade mentioned, Major JUSTICE, I.M.S., the present Sanitary Commissioner for Madras, shewed, in his Annual Report for 1913, that the municipal rate had sunk to 0·3 and the rural rate 0·4; the difference however, as he states, on the population of 42,778,652 represents a waste of 4,277 infants' lives, as a result of lack of sufficiently early resort to vaccination. This question of incidence upon age periods has also been approached by Lieut.-Col. C. E. WILLIAMS, the present Sanitary Commissioner for the Government of Burma, in a very thorough way. The statistics (*vide* Table B *infra*) with which he has been good enough to furnish me, at my request, not only bear out this desirability of protecting life from small-pox at an earlier age than six months, but also support him in arriving at definite conclusions as to the necessity for revaccination. Using statistics furnished by the Health Officer, Rangoon, as to vaccinal condition of 431 attacks (which bear out very fully GAYTON'S contentions as to grade of protection being in proportion to the number of vaccine scars), he states: "I feel strongly the need for revaccination in every person at seven years of age (if primary vaccination was performed under twelve months) and, in all cases, within seven years of primary vaccination. I have no record of a death after revaccination. I consider that a person shewing only one or two successful scars after ten years of age should be classed as "unprotected" for the purposes of the Vaccination Acts. One scar indicates a very doubtful degree of protection, if any." In further remarks upon results in age periods, he states: "As preventing a fatal issue in persons attacked, vaccination performed in infancy or early childhood is thus protective even up to forty years of age, but not noticeably later—as shewn by these statistics."

THE POSITION OF REVACCINATION.

It is not surprising that when vaccination was first practised, in the presence of demonstrations that persons who had been vaccinated were immune to small-pox when inoculated for test purposes with that disease, it should be concluded that they were safe for life. But neither naturally acquired small-pox nor inoculation affords a complete protection to subsequent attacks of small-pox. Slowly but surely, with increased experience in both temperate and tropical climates, the period of estimated necessity for revaccination has been decreased. France now reaps the benefit, so far as males are concerned, not only of infantile vaccination and of revaccination at eleven years, but of this being repeated, by reason of their passing through the conscript army. Although French law now requires both sexes to be vaccinated and revaccinated at eleven and twenty-one respectively, this is not fulfilled rigorously in civil life. The reporting officer, P. PAQUET, who dealt with an outbreak at Beauvais, France, remarks that for five men sixteen women were attacked, and this proportion he regards as normal. In the intensive vaccination campaign in Paris to which I have referred, the inhabitants were invited to undergo vaccination or revaccination as the case may be, if such operation had not been performed within five years. BRUCE LOW quotes Professor KIRCHNER as stating that, during the recent outbreak of small-pox in Germany, it was found that many of the sufferers were middle-aged or elderly. In one thousand attacks, the persons under 30 years of age were 80; over 30 years there were 850, of whom 501 had attained the age of 60. "One sufferer had reached the age of 92. Professor KIRCHNER strongly urges revaccination of all middle-aged persons." Professor SOBERNHEIM sums up this subject by stating that, in 1917, 80 per cent. of the small-pox cases in Berlin were over 40 years of age. (*Lancet*, Jan. 10th, 1920).

The necessity for revaccination was recognised by the Government of Madras in 1894, by their requiring that all candidates for Government employment should undergo vaccination or revaccination as the case might be—unless the evidence was clear that

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either were recent, or that recent small-pox had been undergone. This forms a useful way of educating public opinion in the required direction. A further indirect method to the same end was also adopted by them, namely, the requiring that where schools were offered a grant-in-aid from public funds there should be produced a certificate shewing that the scholars bore vaccination marks. This fitted in well with arrangements by which that Government, as advised by a Sanitary Commissioner of a previous period (the late Surgeon-General BIDIE, C.I.E.), in 1886, formerly sanctioned the now elsewhere largely practised systematic sanitary inspection of schools and colleges, and medical examination of all under instruction attending these, if in any way aided by public funds.

Valuable information is likely to be forthcoming from American practice in the Philippine Islands as to the period within which, in a tropical climate and in tropical subjects, the influence of a primary vaccination may be trusted to. The Americans commenced work in 1898. This, up to recently, has kept at bay epidemic small-pox, which formerly caused about 40,000 deaths per annum. They now have reason to suspect that the limit of duration of immunity has been reached, and the question of general revaccination must be considered.

LIFE-SAVING POWER OF VACCINATION.

This, so far as vaccination *per se* is concerned, must not only depend upon the extent of mass vaccination but upon its efficiency. A partly protected population fulfils the spirit of the Hindu proverb, that "water is not cleared by muttering the name of the clearing nut" (*Strychnos potatorum*). Nevertheless, incomplete efforts are not thrown away. A Parliamentary paper, prepared in 1912, without any differentiation of the various conditions of Provinces, shews that in 1877 (the first year dealt with) the small-pox death-rate for the whole of India was 1.01 per mille of all ages; the percentage of total small-pox deaths under 1 year of age was 21·6; from 1 to 12, 70·65; and over 12 years, 7·99. In 1910 (the last year dealt with) the small-pox death-rate had been reduced to 0·23 per 1,000 of all ages, whilst the percentage of deaths under 1 year was 23·8, and between 1 to 10 years, 34·85; whilst in ages over 10 years the percentage had increased to 41·17. The total life-saving, in a population now amounting to 315,000,000, must, therefore, be very great, although there is exhibited in a marked manner the influence of infantile vaccination in changing age incidence, and the necessity for further practice of revaccination is obvious. Lieut.-Col. JAMES, I.M.S., in his work, *Small-pox and Vaccination in British India*, deals with a mass of statistics, and proves conclusively how vastly mortality from small-pox has declined in British India following the practice of vaccination. The decennial periods from 1868 to 1907, as quoted by him, shew successively, per million of the population of India, the following rates: 1032·3, 771·9, 466·0, 373·9.

VACCINE INSTITUTES.

In the practical work of spreading vaccination amongst large populations in the tropics, difficulties are experienced both in maintenance of the activity of animal vaccine and in its transport to vaccinators. These have been surmounted in various degrees, but there is still much room for improvement, which can only be accomplished by patient research and experiment. To find vaccine stock irrevocably decline in activity and fail to afford immunity to human beings, under modes of cultivation, or a mode of preservation unsuited for tropical climates (though fulfilling their purpose well in temperate climates) are matters of frequent occurrence. Such results must be expected where there is absence of appreciation of the fact that cultivation of vaccine on a large scale cannot reasonably be treated as a mere side-show in the daily work of a medical officer. In no area should cultivation and distribution of preserved vaccine be attempted, unless there exists a Vaccine Institute, constructed with special regard to tropical requirements, equipped with as much care for maintenance of asepticism as in the case of surgical wards, operation rooms and their accessory buildings for human beings, and the working be entrusted to technically educated staffs with no other duty thrust upon them.

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I have, in discussing "types," elected to take case mortality as the test of virulence and not merely their respective incidence upon populations, and have suggested that the Eastern type is that possessed of the highest vigour. So far as figures are at present available to me, it seems that there exists a relation between this virulence and case fatality not only in the unvaccinated, which forms the real comparative test, *but in the degree to which immunity from death can be acquired by vaccination* in those infected with the Western type as compared with those subjected to the Eastern type, as shewn by the following tables. *Consequently it is of special importance that vaccine used in the tropics should be in a high state of activity in areas where this type prevails.*

TABLE A.

Eastern Type (General Rate).

Locality.	Year.	Case Mortality.
Calcutta Hospital	1844	32·3 per cent.
" "	1894	38·8 "
Madras City	1916-17	32·1 "
Rangoon	1896-1916	33·4 "
Bombay City	1914	49·8 "
Straits Settlements... ...	1916-17	33·2 "

Eastern Type (Unvaccinated).

Locality.	Year.	Case Mortality.
Calcutta	1844	66·0 per cent.
"	1894	60·2 "
Madras City	1917	65.74 "
Rangoon (Burma wet zone)	1919	43·2 "
Mandalay (Burma dry zone)	1919	82·5 "
Straits Settlements ...	1916-17	51·6 "

Eastern Type (Vaccinated).

Locality.	Year.	Case Mortality.
Madras City	1916-17	30 per cent.
Rangoon	1919	37·5 "
Mandalay	1919	60 "

Bombay City ... ... 1913 Children under 10 years of age—Vaccinated, 22·5 per cent.; Unvaccinated, 29·5 per cent.

TABLE B.

Eastern Type.

(Small-pox incidence by age-periods in Mandalay City, tabulated by Lieut.-Col. C. E. Williams, Sanitary Commissioner, Government of Burma).

Population by age.	Reported cases of Small-pox.	Ratio per cent. of Population.
0– 5—14,211	... {177, 203} 380 ...	2·67 per cent.
5–10—13,804	... 159 ...	1·15 "
10–15—13,514	... 100 ...	0·71 "
15-20—12,648	... 130 ...	1·03 "
20–40—48,338	... 406 ...	0·84 "
40–60—25,352	... 22 ...	0·08 "
60 & over—10,432	... 1 ...	0·009 "

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TABLE B.—*cont.*

	Cases.	Ratio per 10,000 population.	Percentage case death-rates. Vaccinated.	Unvaccinated.
Under 20 years ...	769 ...	142 ...	59·59 ...	85·56
Over 20 years ...	429 ...	51 ...	60·49 ...	82·09

Case Death-rates.

	Vaccinated.	Unvaccinated.
0–10 ...	67·14 per cent. ...	88·4 per cent.
10–20 ...	49·27 „ ...	55·0 „
20–40 ...	60 „ ...	82·75 „
40–60 ..	84·61 „ ...	77·0 „

Rangoon Hospital.

Total cases = 1,070 (chiefly Hindu coolies over twenty years of age).
Deaths = 293.
Case death-rate = 27·39 per cent.

Vaccinated = 615.
Died = 99.
Case death-rate = 16·10 per cent.

Unvaccinated = 455.
Died = 194.
Case death-rate = 42·63 per cent.

TABLE C.

Western Strain (Unvaccinated).

Locality.	Year.	Case Mortality.
London S.-P. Hospital (General rate) ...	1746-1800 ...	25 per cent. (mixed cases.)*
„ „ (Unvaccinated) ...	1836-51 ...	35·55 per cent.
„ „ „ ...	1863 ...	47·0 „
Provinces, Eng. (McVail) „ ...	1892-95 ...	27·5 „
„ „ „ ...	1902-05 ...	19·3 „

* The introduction to London of small-pox from North Africa gave in the unvaccinated 33·1 per cent.; the general rate is in Tunisia 40·4.

Western Strain (Vaccinated).

(Gayton) Vaccinated with good marks.	All ages.	Case mortality,	3 per cent.
„ imperfect marks.	„	„	9 „
„ no visible marks.	„	„	27 „
Not vaccinated.	„	„	42 „

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DR. ABBOTT'S TABLE.

Places and dates of observation.	Total number of cases observed.	Death-rate per 100 cases.	
		Among the unvaccinated.	Among the vaccinated.
France, 1816-41...	16,397	16·1	1·0
Quebec, 1819-20	—	27·0	1·7
Philadelphia, 1825	140	60·0	—
Canton Vaud, 1825-29	5,838	24·0	2·2
Darkehmen, 1828-29	134	18·8	—
Verona, 1828-39	909	46·6	5·6
Milan, 1830-51 ...	10,240	38·5	7·6
Breslau, 1831-33	220	53·8	2·1
Wurtemberg, 1831½-35½ ...	1,442	27·3	7·1
Carniola, 1834-35	442	16·2	4·4
Vienna Hospital, 1834	360	51·2	12·5
Carinthia, 1834-35	1,626	14·5	0·5
Adriatic, 1835 ..	1,002	15·2	2·8
Lower Austria, 1835	2,287	25·8	11·5
Bohemia, 1835-55	15,640	29·8	5·2
Galicia, 1836 ...	1,059	23·5	5·1
Dalmatia, 1836 ...	723	19·6	8·2
London Small-pox Hospital, 1836-56	9,000	35·0	7·0
Vienna Hospital, 1837-56	6,213	30·0	5·0
Kiel, 1852-53 ...	218	32·0	6·0
Wurtemberg ...	6,258	38·9	3·5
Malta ...	7,570	21·1	4·2
Epidemiological Society Returns ...	4,624	23·0	2·9
Illinois ...	1,931	48·6	6·1

TABLE D.

AMAAS TYPE (GENERAL RATE).

Locality.	Year.	Case Mortality.
South Africa ...	1903 ...	3·8 to 4·3 per cent.

AMAAS STRAIN (GENERAL RATE).

Locality.	Year.	Case Mortality.
America (U.S.A.) ...	1902-15 ...	1·8 per cent.
Australia ...	1913 ...	0·01 "

THE MAINTENANCE OF ACTIVITY OF VACCINE.

Notwithstanding the possession of Vaccine Institutes and technically trained staffs, accidental and unforeseen incidental circumstances not infrequently threaten degeneration, or death, of a vaccine stock. In the absence of NOGUCHI'S method of using testicular vaccine free from extraneous organisms and of exalted activity, and putting aside the temporary expedients of the use of retro-vaccine—a most unsatisfactory mode—equination and the use of rabbits, the most obvious way (and I believe the best) is to get a new stock of variola-vaccine. A few years back, this was as much a matter of controversy as the source of the Nile before the days of Speke and Grant. From the days of JENNER downwards, there has been exercised by members of the profession their proverbial right to differ on the subject. In the present day, lulled by the security obtained by Jennerian prophylaxis, the question of the origin of vaccine or of the true nature of the *causa causans*

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of small-pox is in danger of being indefinitely shelved. CEELY (1839) and several others secured stocks of vaccine by direct small-pox inoculation of bovines, but the unbelievers of the profession hold that, because he had used in certain of his experiments, on different but closely adjacent sites, vaccine and small-pox virus, his success resulted from contamination by the former. Dr. JOHN SHORTT, in 1865, then in charge of the Madras Vaccination Department, performed numerous experiments, and although in certain of the series the effects of too hasty transfer to the human beings are obvious, he evidently obtained positive results; and the Government of the period distinguished itself by being the first to formally sanction the use of variola-vaccine. The Lyons Commission, however, under CHAUVEAU, secured results which put the balance of opinion in favour of it being impossible to transfer from bovines by inoculation of small-pox virus anything but small-pox to the human subject. VOIGT (1881) then made his well-known experiments and secured positive results; but, again, the old cry of the profession was raised against their genuineness, because, as CEELY had done, he used both vaccine and small-pox virus. Thus, the controversy rested till SIMPSON, in 1884, took the matter up, and by direct inoculation of a bovine with small-pox virus secured an indubitable variola-vaccine stock. This was used by the Local Government Board for the vaccination of 1,200 infants without untoward result.

In the course of routine and change of organization, the work of Dr. JOHN SHORTT, in the Madras Presidency, was duly consigned to official oblivion; and when, in 1891, I undertook experiments in the hope of gaining a more vigorous stock than in current use, I was unaware of SIMPSON'S successful work (which was not published till 1897) and also that of Dr. JOHN SHORTT. The water-colours exhibit the results I secured. Since then HIME, COPEMAN, KLEIN and others have been successful, and quite recently Dr. ROSS, in Nairobi, and Dr. ARMSTRONG and associated officers, in New South Wales, have obtained positive results; so that the profession is no longer, on this matter at least, disposed to differ.

Another recognised source of stock is the disease ("cow-pox") as it appears in bovines. In Bengal, MACPHERSON, in 1832, secured a new stock in this way. Owing, however to the fact that Indians use inclusive terms for eruptive diseases, it has been held its origin was rinderpest. In 1894, SIMPSON again came to the rescue, and was able to demonstrate that Bengal cattle (amidst other eruptive diseases) bore cow-pox. In that year, I saw in company with him the vesicles produced from that stock in Calcutta. The point of importance however is not that SIMPSON had vindicated MACPHERSON'S work, but that, for the first time on record, he cultured and sub-cultured the vaccine and maintained its characteristics *in vitro* up to a certain point. The moral surely is that this clue, aided by more modern methods of research, should be vigorously pursued. The disease found naturally in animals and in man would seem a much more appropriate subject for investigation than the more changed form in which variola virus appears in the guise of current vaccine. There is no lack of opportunity for such investigation in the tropics.*

In the absence of knowledge as to the exact conditions under which small-pox inoculation of bovines will afford positive results, it by no means follows that each effort will be successful; there is an element of chance in the matter. Failures in the past have probably been largely due to lack of examination of the animals employed through each stage of the process. Especially it is necessary not to despise apparently trivial indications of skin reaction- indicating variolation. In a series of experiments carried out in my private capacity, in the early part of 1892, I conceived that failures were due to the amount of virus used being insufficient to overcome the resistance of the animal; as now CAMUS has proved in the case of bovines, in which he has secured generalised vaccine. This result, which the Lyons Commission specifically held was impossible in bovines, he

* DUNCAN STEWART found in his search for cow-pox in Bengal a disease amongst cattle, which he regarded as possibly a varicella of bovines. In the water-colour I shew you which is entitled "spurious vaccine," it is possible I chanced upon the same disease in animals. It is noteworthy in reference to varicella, that a German writer (W. STOELZNER) has stated that, under recent war conditions, this disease became aggravated in virulence. Varicella, therefore, would seem capable of varying in virulence as in the case of variola, to which circumstance I have invited attention.

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obtained by a sufficient dose introduced by intravenous injection. My efforts were simply by repeated skin inoculations. I obtained variolation with, at times, so scanty vesiculations as to make me doubtful of their nature. Erythematous eruptions, which under a magnifying glass were found to consist of groups of small papules, a curious effect as of small emboli in the capillaries, and falling of hair resulted. I had obtained variolation without the desired full vesiculation. In due course, I inoculated certain of these animals with a very inferior stock of vaccine. I was surprised to find that the vaccine took, and that the vesicles were finer than the stock warranted, though their maturation instead of being short, as usual with poor vaccine, was now greatly prolonged.

CEELY having failed at the first attempt to inoculate a cow with small-pox virus, inoculated it again at the end of a fortnight, and "deluged" eight punctures with the virus; the result was by the sixth day the animal exhibited characteristic vaccine vesicles.

I shew you a water-colour from nature, by my late wife, of vesicles produced in a variolated calf by degenerate vaccine, within ten days of inoculation with small-pox virus. Instead of these being at their best at the end of 120 hours, they were fully developed at the end of 192 hours. In another instance, the inferior vaccine was inserted at the end of eighteen days from date of small-pox inoculation, and here fine vesicles were developed at the end of 186 hours. There is no doubt in my mind that *by this process it is possible for degenerated vaccine to be rejuvenated.** My experiments were interrupted. In later years I informed Major CHRISTOPHERS, I.M.S., of my views on the subject. CEELY's and VOIGT's mode of producing variola-vaccine naturally occurred to me as bearing on the matter, and I suggested that CEELY's mode was probably correct. In an experiment by Major CHRISTOPHERS, all precautions were taken to prevent the vaccine reaching the variola inoculation site, but he does not guarantee this being above criticism. The result, as shewn in the accompanying photograph, was vesiculation at both sites.

In the light of these results, the work of other experimenters becomes significant. BUIST produced an exceptionally good vaccine scab in a monkey that had been variolated a month previously. COPEMAN used animals simultaneously inoculated with vaccine and variola, and suggests that in "some occult way" the manifestation of the influence of his egg lymph was brought about. SEATON states that M. BOULEY vaccinated a variolated cow and produced regular cow-pox; also that CEELY informed him that "in his variolous experiments on cattle he constantly produced the phenomena described by M. CHAUVEAU, and found subsequent vaccination of these animals, in the great majority of cases successful." The record of experiments by Dr. ARMSTRONG and associated officers concerning the "mild type" of small-pox in New South Wales, contains an instance where a calf having been subjected to variolation with a "slight, somewhat doubtful take" was vaccinated on the eleventh day, and afforded a "good typical take."

The connection with BRYCE's test and the various conditions stated would seem possible. Dr. JOHN SHORTT, whose experiments I have alluded to, states: "In one instance where four insertions of small-pox matter from cow failed, reinsertion of genuine vaccine matter, as late as the seventh day after primary operation, caused the original small-pox insertions to vivify and run their regular course. This is an interesting feature in this case, as the secondary insertions only shewed pimple-like formations, and the cicatrices were also equally satisfactory."

I think, therefore, that one may finally put away the idea that CEELY owed the success of his small-pox inoculations to contamination by vaccine and that, on the opposite, both he and VOIGT used a sound and more certain method of getting variola-vaccine stock than when small-pox virus alone is used. Further, I think there are good grounds for believing that where an attempt to get variola-vaccine has been made, with simple variolation without vesiculation resulting, there is still open the mode of rejuvenation of vaccine, by use of the animal hitherto regarded as of no further use.

Again, the successful procuring by CAMUS of generalized vaccinia by using intravenous

* *British Medical Journal*, November 7th, 1896, p. 1377.

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injection shews that the theory on which I started the series of experiments I have alluded to, namely, that vesiculation is the full expression of submission of an animal to small-pox virus is sound; whilst the last mentioned experiment by CEELY would go to prove that though vaccine precipitates that result in an already variolated animal, repeated small-pox virus inoculation will also accomplish it. Indeed, having regard to the upsetting of CHAUVEAU's doctrine *re* bovines by CAMUS, it would seem in future that the certainty of the sufficiency of the dose of small-pox to obtain vesiculation on bovines might best be secured by intravenous injection.*

But I do not think the facts I have gathered together, though in themselves crude, need fail in having further possible useful application. In 1892, SIMPSON stated before the Clinical Society of Calcutta, that he thought small-pox was of dual nature. Since then (1908) PROWAZEK and BEAUREPAIRE ARAGAO have suggested symbiosis as a probable factor in vaccine production. It seems to me quite possible that the final determination of vesiculation in a calf submitted to a small dose of variola, so small as to be capable only of expending its force solely upon the internal organs, might well be the result of skin disturbance caused by vaccine virus known, as NOGUCHI states, to prefer pavement epithelium. Finally, if the Chinese successfully cause communication of small-pox by blowing scabs into the nostrils, may not the dual nature or symbiosis be due to an ultra-microscopic organism which, as suggested in influenza, may require a symbiosis for full exhibition of virulence; and, in any case, why should not the naso-pharyngeal passages be searched for the hypothetical microbe—as an important receiving area of infective matter.

VACCINATION DEPARTMENTS.

Having arranged for the out-turn of vaccine in an active condition, and of a sufficiency calculated with reference to the requirements both of primary vaccination and revaccination, as well as for emergencies for epidemics, the next step in small-pox prevention would be to see that the vaccine produced was utilised to the best advantage. This can only be secured by determining to utilise well educated and reasonably paid men. It is hopeless in most tropical areas to expect that none but fully qualified medical men should undertake vaccination duty. On the other hand, a poor class of lay vaccinators not only means poor out-turn of work, but in rural areas, where supervision is difficult, falsification of returns in the interest of laziness results. It would be impossible to form a standard plan of organisation. I think, however, that the framework of a scheme I proposed for the Madras Presidency may give some idea of the character of personnel desirable where medical men cannot be afforded. This scheme has been in operation "on trial" in three districts since 1905, and is now (doubtless with modifications by my successor, Major JUSTICE, I.M.S., resulting from experience) about to be used throughout the Presidency. The Madras Presidency is rich in sanitary inspectors. To become an assistant sanitary inspector a man must have passed the matriculation examination of the Madras University, or some higher examination. He then undergoes a course of theoretical and practical training in hygiene, physiology, and bacteriological demonstrations during six months. To become a sanitary inspector he has to undergo a further course of five months in minor sanitary engineering. During his further career he undergoes periodical examinations, and special training at the end of five years to make sure he does not lag behind the age. Such men make a good class of vaccinators. For this purpose, after qualifying as assistant sanitary inspectors, they are required to undergo a three-months' course of training in the following subjects:—preservation and bacteriology of vaccine, and the science of asepticism; human and animal vaccination (theoretical); official routine and law concerning vaccination; common skin and other diseases of man (theoretical); human and animal vaccination (practical); common skin and other diseases of man (practical); common skin and other diseases of

* A. D., one of the writers in the *Daily Review of the Foreign Press* (Medical Supplement) suggests as to the human being—"The method of intravenous inoculation, once the dose established, would result in no such prolonged disability nor discomfort, and would give, in all probability, an immunity lasting for a very long period."

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animals (theoretical). From these men, by seniority and selection, are obtained their immediate superiors, deputy inspectors of vaccination. The staff thus formed is under the supervision of a special medical officer, who fills the post of sanitary assistant to the district medical officer, and the whole department is subject to inspection by the Government Sanitary Commissioner and his deputies. In whatever area used, I think the class of men indigenous to the country concerned, such as I have sketched, represents the minimum of technical labour that should be trusted to. The utilisation of a staff of this character may of course be limited by the fact that advance can only be hoped for in certain parts of the tropics by allotting a reasonable area to the staff, and trusting to various means of persuasion and inducement to cause the people to accept vaccination. But in the more advanced areas, where compulsion can be employed, as will be the case in the Madras Presidency, the next step is to require compulsory registration of births and deaths, accompanied by compulsory vaccination. On this basis, it becomes a simple matter to work out the necessary rulings for the complete working of the desired department.

Sanitary Organisation against Small-Pox.

If sanitary organisation is to be relied on, the staff certainly should not be allowed to think they have done well by comparing results with a group of areas where attacks have been one per mille (!) as suggested by Dr. Millard. Staffs should be so organised. and in proportion to the area under their charge, that they should possess instant intelligence of attacks and prevent spread. With a vaccinated population the possibility of tardily obtained evidence leading to spread may be more readily combated than if this reasonable precaution has been neglected; hence, the desirability of the first line of defence being vaccination, with sanitation as a reserve that is available the moment danger is threatened.

I need not go into the pros and cons of action necessary in localities; but there is a branch of sanitary organisation which demands notice. The periodicity of epidemics of small-pox has a relation to exhaustion of susceptible material and, in the presence of a pandemic disease, no country can be said to be free from fear of invasion. Hence, there are conditions under which it should be possible for one country to rule that persons from another country should not be allowed to enter it, unless freedom from certain communicable diseases has been ascertained. There, of course, must be wide limitations to such rulings; I refer solely to the principle. Aliens seeking labour in tropical countries form a class to which this principle is peculiarly applicable, and a reasonable safeguard certainly is that recent vaccination has been undergone by individuals concerned. For example, along the border of Mexico, where small-pox is rife, compulsory vaccination of all persons entering America at certain points is required. But it is upon port health staffs that England and many other countries largely depend for defence against invasion by small-pox, as well as other communicable diseases. The Public Health Service of America furnishes the type of what is requisite. By placing special officers at the chief foreign ports and, in the minor, trusting to Consuls, the American health authorities maintain touch of diseases throughout the world. It will take a long time for our Empire to "waken up" sufficiently to see the necessity for this. In the meantime, few of us remember how much we are indebted for protection from infectious diseases to the devoted labour of our Port Health Officers. Some idea of possibilities is furnished by the following rough details:—In the Port of London, between 1906 and 1914, the mean annual average cases of small-pox dealt with was 23·1. During 1919, ships either brought cases, or had before arrival landed cases, which had been infected in Chinese waters (twenty cases), Jerusalem (seven cases), Rangoon, Calcutta, Singapore, Alexandria, Oporto and Russia. Recently, in the Manchester Port, four cases were landed, which had been infected in Egypt; in January, Glasgow received three cases from Lisbon. In the Port of Liverpool, between 1914 and 1918, small-pox cases were received from Oporto, Baltimore (?), Archangel, Rangoon, Cetto and Jaffa. One of our most experienced Port Health Officers, Professor Hope, in a paper contained in the *Journal of State Medicine* for April, 1919, in a forcible manner, calls attention to the error of ignoring the necessity

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for the pandemic disease of small-pox being brought within the scope of an International Convention. That such an essential proceeding has been neglected up to date can only be accounted for by the existence of a false sense of security which yearly has less reason to justify it, in view of fast increasing exemptions of "conscientious objectors" in all parts of the world. He states as follows:—

> "The work will be carried on at a higher level of efficiency, and many new phases of development will appear in the near future. The health administration of ports all over the world will be considered of first importance, not only in safeguarding the health of the nations, of which they are the first line of defence, but also from the international standpoint. There can be no doubt that international co-operation to prevent disease of all kinds will be more in evidence than in the past. Already International Conventions have arranged many matters in regard to plague and cholera. Why should such conventions not deal with small-pox, enteric fever, and other infections which are now endemic in certain regions of the world? Much good would result from a consideration of this subject."

Discussion.

The President: Colonel King has given a masterly exposition on the subject of small-pox and its prevention. Those who know him, and are acquainted with his work, would expect such an exposition. He has devoted many years of his professional life to endeavouring to solve some of the problems connected with small-pox and vaccination. He got into very serious trouble, when a young man, for carrying out certain experiments on variola and vaccine which were disapproved of by his superior officer, and for the time being he lost his position. However, later on, he was reinstated, and succeeded in placing the preparation, storage and transport of vaccine lymph in the tropics on a scientific basis and maintaining its efficiency. It was at that time that he introduced the system of mixing the vaccine, not with glycerine—which he found unsuitable for storage and transport in the tropics—but with pure lanoline, a method that has been used with advantage in many parts of the tropics, and has been found of the greatest value.

There is no need of Colonel King apologising for bringing the subject of small-pox before this Society, because all those who know the tropics are aware what a scourge small-pox is there. He has demonstrated that small-pox is more destructive in tropical regions than in colder climates. And it is very far from dying out. The dying-out theory is raised by those who confine their attention to small-pox in such countries as England. If they would travel they would soon change their mind.

The map which has been exhibited shews the wide distribution of small-pox throughout the world, and the great difference it presents in virulence according to its geographical position. The type Colonel King speaks of as the Eastern—which, I think, most authorities believe to be the most virulent type, and which provides the chief parental stock—ranges between a case mortality of 60 and 80 per cent. among the unprotected. In other parts of the world this is reduced to 40 and 45 per cent. among the unprotected. In the particular "barred region," which the author speaks of, it is as low as 20 to 30 per cent. among the unprotected. This means a reduction in the case mortality of at least one half in certain parts of Europe compared with the case mortality in the East. This is very much like what occurs in some other infectious diseases. The European seems to resist these infections much more than the inhabitants of the East.

With regard to the dying-out idea, I had the opportunity of seeing a report, not long ago, of an outbreak of small-pox in Togoland, which, as you know, once belonged to the Germans. There were nearly 5,000 deaths in a district with 60,000 inhabitants. The inhabitants had been, according to the German views, thoroughly vaccinated. The considerable doubt I had whether this was the case was owing to the statement

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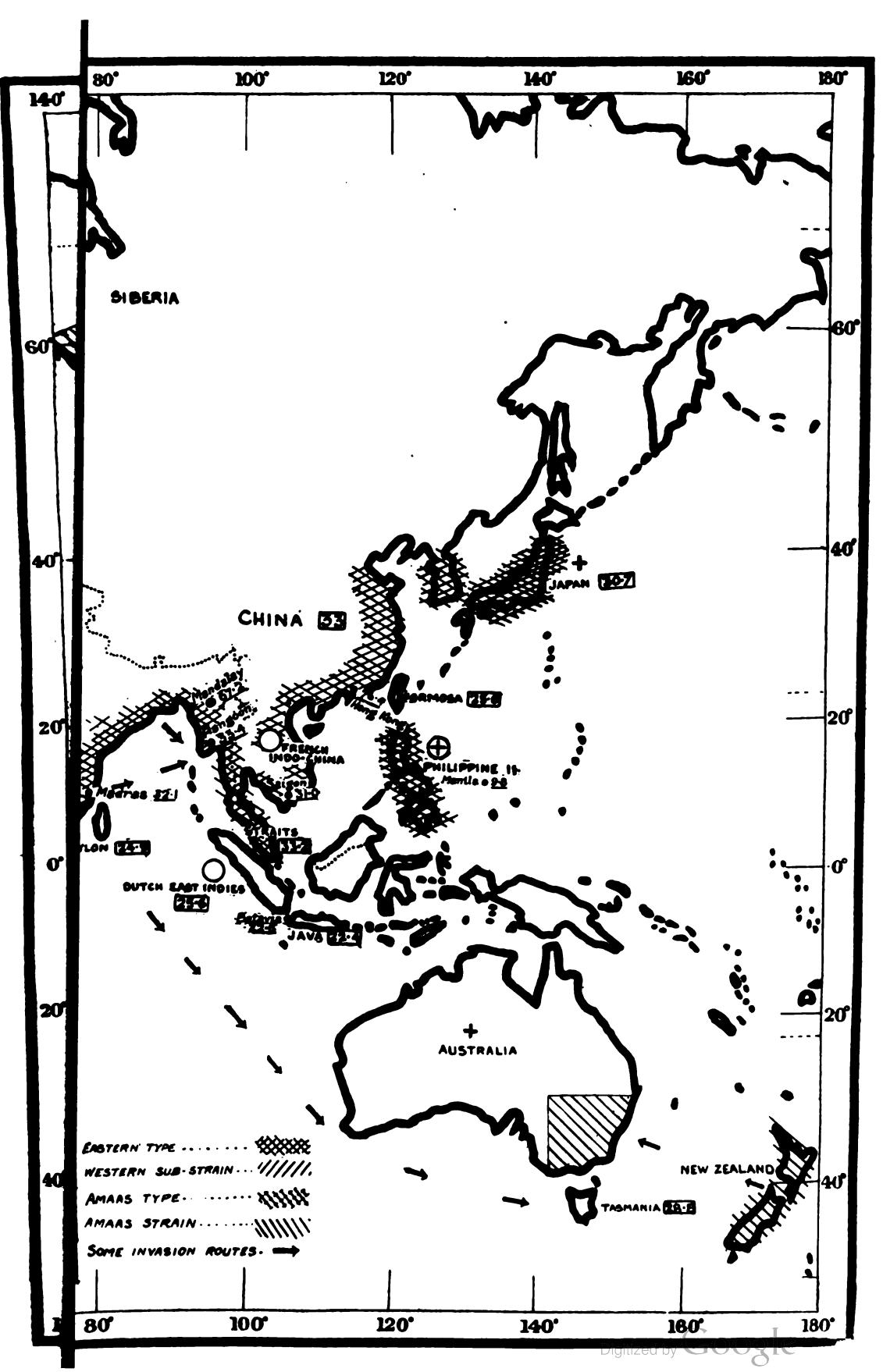

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that the vaccination took best during the rains. The experience of most people who have been practically connected with vaccination in the tropics, is that it is very difficult to keep up effective vaccination during the rains in the tropics, for what reason one is hardly prepared to say. In Calcutta, I took the opportunity, when we had the epidemic of 1894, of analysing the respective effects of inoculation and vaccination on the case mortality, and comparing it with case mortality in the unprotected. I found that among the vaccinated under five years of age there was no case mortality, but between five and twenty years of age it rose to 3 per cent., while from twenty upwards it increased to 10·6 per cent. These were the percentages for the vaccinated. When it came to inoculation, there was among them no case mortality up to twenty years, but at later ages the mortality was 19 per cent. Now, referring to those who were unprotected, the case mortality under five years of age was 50 per cent., and over 75 per cent. under one year of age. Between ten and twenty years of age it came down to very nearly 30 per cent. After twenty, however, it reached over 65 per cent. That is a useful comparison.

The type mentioned in connection with South Africa, America and Australia seems to have been extraordinarily mild, the case mortality ranging from 7 per cent. to ·7 per cent. It is possible that the mild and the virulent types may be associated in the same locality. I do not think that the mild and virulent types owe their special properties to any marked change that has been effected in their passage through man, but I am inclined to think the fixing of the type, as to virulence or mildness, is brought about by passage through some of the lower animals. I am convinced it is in the lower animals we are likely to find a solution of some of these problems. We know that horse-pox and cow-pox are produced, at all events artificially, by means of inoculating with variola, and the change is enormous: the small-pox has lost its infectivity, yet it retains in a fixed form its immunising powers. Seeing that this great change can be effected by passage through the horse and the cow, it is not unlikely that some lower animals will be found which can raise both the virulence and infectivity of small-pox. I feel certain, from many observations in India, that many of the diseases which pass under the name of "cattle plague," "rinderpest," and such like terms, although they present very different symptoms from those of small-pox in man, should not be passed over as having no connection. You have an illustration of dissimilarity in the infection of rabies. Rabies is very different in the dog and some other animals from what it is in man. Bearing this in mind, it is always suggestive to look in that direction. I think some of our younger members need not be under the impression they would be wasting time or opportunity if they were to follow up this idea; I am convinced it would be a fruitful line of research. My object when endeavouring to cultivate vaccine was not wholly in regard to the cultivation of vaccine *in vitro;* it was with the object of seeing whether the microbe—whatever it might be—could not be found in some other animals which, apparently, had not this disease. By this means I hoped to find the animal or animals which raised the virulence and infectivity of small-pox and conveyed it to man, and by dealing with the animals to prevent its transmission to man.

Professor ALDO CASTELLANI: I would like, first, to congratulate Colonel KING on his most interesting paper—the paper of a real expert.

I may, perhaps, be allowed to say a few words on two points, one in connection with the differential diagnosis of small-pox, the other in reference to the treatment of the disease.

We all know how difficult it is, in certain cases, to make a diagnosis between small-pox and chicken-pox. It is still more difficult—in fact, I believe, clinically it is impossible—to differentiate between true small-pox and alastrim. But there is another condition, a rare one, from which small-pox must be differentiated—a condition which I described a number of years ago and called "vesicular fever."

This condition generally begins abruptly. The patient complains of feeling very feverish and often of severe pain along a certain nerve, frequently the supraorbital. After two or three days, vesicles appear, and one naturally thinks that it is a case of herpes zoster. Two or three days later, however, vesicles develop all over the body: they are smallish,

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rounded, flat, somtimes umbilicated. The contents of the vesicles are, at first, clear, but after a few days become purulent. They heal, leaving a small scar. The condition is certainly different from true small-pox, because after an attack of vesicular fever JENNER's vaccination is positive. It is also different from alastrim, because the pustulation is much less abundant, and the pustules are much smaller in size. Moreover, in alastrim the peculiar neuralgic pains have not been described.

As regards chicken-pox, it differs from this condition as the vesicles are much larger, and there is a good deal of crust formation.

A word with reference to treatment: I have always found it useful to place the patient when possible in a room with red light, a procedure first introduced by FINSEN. Another measure I have often found useful is painting, once or twice a day, all the papules, and later the pustules, with tinct. iodine or with nitrate of silver dissolved in spirit of ether. If these measures are regularly carried out, in a number of cases the scarring is reduced to a minimum.

Major NORMAN WHITE: None of us who have worked in India are unmindful of the great services Colonel KING has rendered with regard to vaccination, and the paper he has just read places us still further in his debt. Those who have been engaged in health work in India in recent years realize that Colonel KING's remarks about the activities of the "exponents of anti-vaccination" are only too well founded. Whatever support can be advanced for views such as those of Dr. MILLARD in the conditions pertaining in this country, such views transferred to India, in the manner in which they are transferred, are capable of doing, and are doing, a great deal of harm. Colonel KING's able summary of the situation should be of the greatest assistance to those whose duty it is to combat such views.

I think no chapter in the history of health administration in India affords more grounds for satisfaction than does that relating to the prevention of small-pox. The results have indeed been surprising. The reason of this is not far to seek. At the present time certainly not less than 50 per cent. of the total expenditure on disease prevention in rural India, which contains more than 90 per cent. of the total population, is devoted to vaccination, and yet small-pox to-day is responsible for less than one per cent. of the total mortality in such areas. I do not for a moment advocate a lessened expenditure on vaccination, but if only those responsible would see in the history of small-pox prevention the urgent necessity for dealing with other serious communicable diseases on similar broad lines, who can doubt that the results achieved would be commensurate with the expenditure involved! I am not of course advocating a separate staff to deal with each of such diseases; such a tendency has perhaps been too much in evidence in the past, and not only in India. Rather would I see vaccination entrusted to a staff sufficiently well trained to deal with disease prevention in general. Further discussion of such matters would lead too far from the subject of the paper, to which I have listened with much interest and profit.

Colonel ANDREW BALFOUR: I do not feel competent to discuss this very erudite paper: some of the problems with which it deals are difficult and intricate, and can only be properly handled by experts, such as Colonel KING and our PRESIDENT, both of whom have made a close study of the subject.

But there are one or two points to which I might refer. When I was in British East Africa, early in 1918, I was asked by the Colonial Office to proceed to Uganda and furnish a report on the failure of the lymph manufactured in Nairobi It was then I discovered how very little is known of the subject from a scientific standpoint as far as work in Africa is concerned. Still, it proved a very interesting enquiry, because of the numerous factors involved. There is no doubt the lymph was not successful in Uganda: it brought vaccination into disrepute, and, wherever possible, the natives were avoiding vaccination. Among the conditions causing this failure was that of transport, and transport of lymph in tropical countries is a matter which requires very careful consideration, because, as we know, the effect of heat on vaccine lymph is greatly to diminish, and even abolish, its potency. Various methods have been tried to secure the effective transport of lymph,

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but it would take too long now to go into them. At present interesting experiments are being carried out in Uganda, as regards the transport of lymph. They have been undertaken largely at the instance of Professor SIMPSON. Special flasks for the purpose have been devised, somewhat on the principle of thermos flasks.

But there were also other factors: for instance, the question of faulty technique. Native vaccinators had not been sufficiently trained, or they had forgotten their training, and all manner of errors had been made in vaccinating. Too much antiseptic was sometimes applied to the skin, and mistakes were sometimes made in connection with opening the capillary lymph tubes. Sometimes, at least in East Africa, bottles were employed, in place of tubes, and contamination took place. Again, patients were now and then allowed to stand in the sun after they had been vaccinated, and, naturally, this militated against success.

Another question of considerable interest is that of delayed vaccination—delay in the appearance of the vesicle. It is a point which has not been properly cleared up, and I would like to ask Colonel KING what his views are in regard to delayed vaccination.

Apart, however, from all these faults, there was the fact that something had gone wrong with the strain in Nairobi: it had become, in some way, weakened. I remember a recommendation was made to heighten its virulence by passing it through monkeys (*Cercopithecus*). This was the method in vogue at Dar-es-Salaam, and the results were said to be good.

But how do you know you have got a good result from vaccination? In the tropics the production of a good vesicle, even the classical "pearl on the rose-leaf," is apparently not sufficient proof: there may be good vesicles, and yet the protection afforded may not be adequate. What we require is some simple, easily-applied test in order that we be sure as to the potency of our lymph. Such a test will be difficult to obtain until the necessary laboratory facilities in properly equipped vaccine establishments are provided in tropical countries. I am glad to say that Colonel KING has prepared for the Colonial Office a very important and useful memorandum on the question of such Institutes and their work. It will be distributed throughout the African Colonies, and I have no doubt it will be most helpful. This important question is not adequately dealt with in our English literature, and it is at present difficult to get information on the problems which confront the workers in the tropics.

There are difficulties in the tropics, but there seem to be difficulties at home also, because in Egypt, during the war, there appeared to be evidence shewing that some of the medical officers did not know how to vaccinate properly, and it is a matter for serious reflection that in 1916-17, eighteen British soldiers died of small-pox in Egypt.

Professor SIMPSON has told us of the death of an anti-vaccinationist from that disease. I remember a case in which the Censor found in a letter from a soldier who had been vaccinated, a boast as to how he had evaded vaccination by licking off the lymph after its injection. That man died of small-pox at a later date.

Dr. TODD, the head of the Hygiene Institute in Cairo, finds that water buffaloes are better than Egyptian cattle as furnishers of vaccine lymph. I have discussed this matter with Colonel KING, and he does not agree with Dr. TODD's view. Opinions vary considerably as to the best methods of heightening the virulence of the vaccine, and as to the best types of animal from which it is to be derived. Hence there is still need for research into the whole subject, at least as far as the tropics are concerned

I was very interested to hear what Colonel KING said about the virus in the naso pharynx. Recently, I came across a reference in the *Journal of the American Medical Association*, which I will read to you:—

> "SOBERNHEIM* expatiates on the importance of an early diagnosis of small-pox. The extremely fine granula found by PASCHEN in all the small-pox cases he examined requires great skill in the technique for its discovery, but it most certainly seems to stand in some relation to small-pox, as also GUARNIERI'S cell inclusions which can be refound in the rabbit cornea inoculated with pustule contents. This test also requires a complicated

* *Small-Pox and Vaccination.* By G. SOBERNHEIM. *Correspondenz-Blatt fur Schweizer Aerzte*, Basel. Dec. 4. 1919. *49*, No. 49. (Reviewed in *Journal of the Amer. Med. Assoc.*, Jan. 24, 1920.)

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technique, but PAUL'S method is simple and rapidly instructive. With this, the scarified rabbit cornea is inoculated with contents of the suspected pustule or with secretions from nose or throat, and in thirty-six to forty-eight hours there is a macroscopic reaction, the cornea around remaining normal while minute elevations can be seen at the infected points. If the eyeball is placed for a short time in mercuric chlorid-alcohol, the cornea turns grey while the minute swellings show chalky white. This kind of reaction is seen only with small-pox or vaccine virus, but negative findings are not conclusive. With this and other tests, it has recently been demonstrated, he adds, that the virus is found in the nose and throat early in the disease, and that the disease can be transmitted by droplets, but no evidence of healthy carriers has been discovered as yet."

I cannot express any opinion on this matter, but hope that someone here may be able to discuss these important findings.

Colonel W. G. KING: In the case of delayed vaccine results, I should say, you are simply dealing with a vaccine which has departed from type. In a Vaccine Institute no such thing should occur. Occasionally you may get a variation from type, either in the human being or in the animal, in which vesiculation is delayed. But if this characteristic persists, it becomes evident the vaccine has departed from the type which you suppose it follows. Usually, however, in the tropics, a delayed vaccine means a strong vaccine, though it is always possible a strain possessed of little vitality might elect to fizzle out, as Colonel BALFOUR describes. For example, first cultivation from variola vaccine is decidedly delayed; it takes some time before it conforms to vesiculation fully at the 120th hour. As a rule, a weak vaccine shews itself by precipitating vesiculation. I remember, according to a late report, in the case of the Accra laboratory, they were dealing with an inferior vaccine which matured at the 72nd hour, shewing a hastened result. The rule in every institute must be to have normal type and maintain it.

With regard to the mode of ascertaining whether it has reached a certain virulence or not. That has been met by HENSEVAL and CONVENT by the use of dilutions. NOGUCHI cultivates his testicular vaccine up to a virulence which will give confluent vesiculation on the back of a rabbit in a dilution of 1 in 10,000. HENSEVAL and CONVENT believe that a vaccine which will give, in a dilution of 1 in 500, a vesiculation of that sort is sufficient. As I mentioned in my paper, in dealing especially with the Eastern type of small-pox, the actual virulence of vaccine should be ascertained. It was not until I myself went over all these figures, to ascertain whether strains and types existed that I realised that the Eastern type of small-pox is of the deadly virulence it is. The lesson of the figures I have set before you is, that we must be prepared to deal with the Eastern type by means of a very special vaccine. Its virulence not only exhibits itself as deadly in the unvaccinated, but also in the vaccinated. For example, there is no comparison between the results of vaccination in India and those in this country: there is about 20 per cent. difference in the death-rate. Therefore, you must have a special vaccine to combat this Eastern form, and it should be kept up to strength by special methods of cultivation, such as NOGUCHI inaugurated, or be frequently renewed by going back to stock variola vaccine.

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THE CAUSES OF DEATH FROM MALARIA IN PALESTINE—A STUDY IN CELLULAR PATHOLOGY.

BY

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INTRODUCTORY.

During the recent successful operations in Palestine, the high death-rate and clinical severity of malignant malaria (*Plasmodium falciparum*) amongst British Troops, Egyptian Labour Corps, and Turkish Prisoners, constituted the predominant clinical and pathological experience of the campaign.

An analysis of the actual cause of death, as revealed at autopsy, is given in the accompanying table. (Table I).

In all these cases malarial parasites were demonstrated, either in the peripheral blood during life, or in the internal organs at autopsy. Of these eighty cases, seventy-three were found to be malignant malaria, while six were benign, and one a quartan infection; but in the latter benign infections it is probable that the malarial element played but a contributory part in the fatal issue. As a direct cause of death, and as a most potent predisposing cause, the malignant tertian parasite was undoubtedly supreme.

ANALYSIS OF FATAL CASES OF MALARIA.

TABLE I.

Nationality.	Total Cases.	Uncomplicated Malignant Malaria.	Bronchopneumonia.	Bacillary Dysentery.	General Sepsis.
Egyptians	11	2	4	3	2
Turks	47	10	27	10	0
British and Australian	22	1	16	2	3

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The most striking feature of this table, which is compiled from cases in base hospitals, was the comparatively small number dying from malaria uncomplicated by any other infection, that is, only 16·4 per cent., while in the remainder, 83·6 per cent., death was due to some secondary infection. The figures should be contrasted with those obtained nearer the source of malarial infection in the scene of military operations, but even here BAHR's figures, in an analysis of sixty-seven fatal cases, shew that 47·6 per cent. were due to secondary infections.

Broncho-Pneumonia.—The most frequent complication was undoubtedly broncho-pneumonia; this occurred in 59 per cent. of the total number of deaths.

The pneumonic consolidation appeared to be due to a bacterial infection and not to any mechanical obstruction of the pulmonary capillaries by the sporulating malignant parasites; in other words, the malarial element played no part in the actual pneumonic process. Of micro-organisms, the streptococcus, pneumococcus, and a gram-positive diplostreptococcus were most frequently demonstrated; *Bacillus influenzæ* itself was found in small numbers; occasionally other gram-negative bacilli and staphylococci were present.

Bacillary Dysentery.—The second most commonly imposed infection was bacillary dysentery (*B. dysenteriæ*, SHIGA, isolated from the stools); this occurred in 17·7 per cent. of the total number of cases. Occasionally acute bacillary dysentery killed in a few days, but in others it ran a subacute course, and the patients died of exhaustion, having failed entirely to respond to antiserum treatment.

Septicæmia.—In five cases death was due to a septicæmia, secondary to some primary focus. One resulted from the spread of infection from an osteomyelitis of staphylococcal origin (*S. aureus*).

In three fatal malignant cases the *Staphylococcus pyogenes aureus* was actually isolated from the bloodstream during life by hæmoculture. The primary focus was apparently so unimportant that in a normal subject one would never have suspected that generalised infection would supervene. In two of them, pyæmic abscesses were actually scattered throughout the lungs.

It is interesting to note that in four malarial patients, who ultimately recovered after operation, an apparently cryptogenic purulent arthritis supervened. In three cases the staphylococcus was responsible and in one the gonococcus.

Blackwater Fever.—There was one remarkable generalized infection with *B. perfringens*, in which coincidentally and apparently intimately connected with this septicæmia, a typical blackwater fever attack with acute hæmolysia supervened eighteen hours before death.

This case illustrates the potentiality of a malignant malaria to the condition known as blackwater fever.

Our observations also substantiate the occurrence of a *transient* and *slight* hæmoglobinuria in cases of quite average severity in whom a clinical picture was in no way suggestive of blackwater fever, an experience which tallies with a published statement of other observers in Salonica.

THE EFFECT OF QUININE ON MALARIAL PARASITES AS DETERMINED AT AUTOPSY.

In cases of uncomplicated malignant malaria coming to autopsy after inadequate quinine treatment, parasites were readily demonstrated in the internal organs, especially in the spleen in which also malarial pigment was recognisable macroscopically by its tarry appearance and diffluent state. Subsequently, of course, this was verified by direct microscopic examination. In nearly all these cases quinine administration had been either deficient in amount or delayed until too late. On the other hand, in cases of malaria dying of some secondary infection who had received numerous injections, it was otherwise.

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In a series of twenty such cases who had received at least three injections of grs. 12 quinine bihydrochloride no parasites at all were present in the spleen smears, while a large amount of malarial pigment was present in sixteen of them. In the four remaining ones, the primary malarial attack had so far anteceded the cause of death, that the characteristic pigment had disappeared from the spleen entirely. These observations on the fate of the parasite after three intramuscular injections on consecutive days, the disposal of the malarial pigment and the intimate connection between these acute cases and the condition known as blackwater fever were made synchronously with, and quite independently of, those by Major MANSON-BAHR, D.S.O., R.A.M.C., and are in agreement with his findings.

These observations suggested that malarial infections of the malignant type are specially prone to secondary complications and that when once these have manifested themselves the prognosis is grave indeed. In order to investigate this aspect of the pathology of pernicious malaria, systematic examinations of the blood and bone marrow were made both in complicated and uncomplicated cases.

OBSERVATIONS ON THE CELLULAR PATHOLOGY OF THE BLOOD IN MALIGNANT MALARIA IN THE FIRST TWO MONTHS OF THE DISEASE.

1. *Total leucocyte count.*—The outstanding feature of the total count was the almost constant leucopenia, both in the *pyrexial*, as well as the *apyrexial* period. In forty cases examined during the *pyrexial* period, the average total count was 5,200 leucocytes per c.mm.

In twenty-one cases examined during the *apyrexial* period the total count was a similar one, viz 5,700 per c.mm. In those complicated by broncho-pneumonia the leucocyte response was often very feeble, frequently the count not rising above 12,000 per c.mm.; while in others *no response occurred at all* and the diminution persisted in spite of the stimulus of a secondary septic infection.

These observations are novel, in that they do not agree with those of other workers or the leucocytic variation in malaria. The probability is that the latter were dealing with *relapses* of both benign and malignant infections, while the present deal with either fatal or early and severe primary infection of a virulent type.

2. *The differential leucocyte count.*—The average differential count in fifty uncomplicated cases in British troops was as follows.

Polymorphonuclear leucocytes	53·34	per cent.
Lymphocytes	25·54	,,
Large mononuclears and transitionals	21·03	,,
Basophils	·08	,,
Eosinophiles	·01	,,

The dominant feature to note was an increase in the mononuclear and transitional cells; this is known to occur in other protozoal diseases with splenomegaly, and it is probable that these cells have their origin in the endothelial and pulp cells of the spleen. The more chronic the cases, especially those with crescents, the higher was this mononuclear count found to be. There was also found a definite decrease, both relative and absolute, of the neutrophile leucocytes, while the eosinophiles were almost entirely absent.

3. *The red blood corpuscles.*—The blood pictures of the *average* case, four to eight weeks after infection, shews a polychromatophilia with a hæmoglobin percentage of 60, red cells 3,500,000, and a colour index of ·8.

The virulent infections shewed a very definite and severe anæmia with polychromatophilia, anisocytosis, and poikilocytosis.

Megalocytes were frequently observed, while *megaloblasts* were present in a large proportion of cases, and *normoblasts* occasionally, thus simulating very closely the blood picture of idiopathic pernicious anæmia.

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There were also clinical resemblances between these two diseases, for instance, retinal hæmorrhages and cardiac murmurs of hæmic origin.

TABLE II.

No.	Type of Infection.	Hæmoglobin.	Leucocyte Count.	Red Cells.	Colour Index.	Spleen.	Clinical.
1	M.T. Rings	20 per cent.	4,500	1,400,000	·7	+ + +	Jaundice.
2	,,	10 ,,	—	1,830,000	·3	+ + +	——
3	,,	20 ,,	7,500	2,050,000	·6	+ + +	Nephritis and retinal hæmorrhage.
4	,,	30 ,,	5,000	2,450,000	·6	—	——
5	,,	15 ,,	5,000	1,100,000	·7	+ + +	Retinal hæmorrhage.
6	,,	15 ,,	2,800	1,110,000	·7	+ + +	,, ,,
7	,,	30 ,,	12,000	2,040,000	·75	+ + +	Intestinal hæmorrhage
8	,,	20 ,,	5,800	—	—	+ + +	Jaundice.
9	,,	30 ,,	4,400	2,050,000	·7	+ + +	——
10	,,	50 ,,	7,000	3,000,000	·8	+ + +	Transient hæmoglobinuria.

With reference to this table, it must be understood that the figures refer only to the most pernicious cases arriving at the base, and not to those of average severity, though all shewed a definite anæmia.

Observations on the Cellular Pathology of the Bone Marrow in Fatal Cases of Malaria.

The specimens of bone marrow were obtained from fatal cases of complicated and uncomplicated malaria in Turkish Prisoners of War and in the Egyptian Labour Corps. The smears were taken from the middle third of one of the lower ribs as being the site of election.

Smears were made on cover glasses, fixed in Schaudinn's fluid and stained with Gauducheau's modification of Romanowsky's stain.

In contrast with normal, the acute malaria bone marrow smears shewed the following noteworthy points: —

1. There was a most remarkable erythroblastic reaction so that in some instances the nucleated red cells amounted to fifty to sixty per cent. of the cells in one microscopic field. That an erythroblastic response of this nature occurred in the centre of the rib is some indication of the stimulus given to red cell production.

2. A great number of the cells were of the more primitive *megaloblastic* type with faintly acidophile cytoplasm and actively dividing nuclei.

3. Large phagocytic cells were present with ingested red corpuscles, leucocytes, malarial pigment, malarial parasites and much chromatoid matter of doubtful origin. There was no evidence that the phagocytic power of these cells had in any been inhibited.

Possibly their undoubted avidity for apparently undamaged red cells in malignant malaria represents a safeguarding mechanism against intravascular hæmolysis. More research is required upon this point. In this the explanation of blackwater fever may be ultimately found.

4. Parasites were present in all cases thus examined, and there was also a large amount of the characteristic pigment which had been ingested by the phagocytes.

5. The myelocytes were relatively scarce and shewed no mitotic figures which are so typical of these cells in normal marrow.

Thus, in contrast to the red cells, there is a complete absence of a *leucoblastic response*. This picture affords a remarkable contrast to that of relapsing fever in which disease there is a very marked leucoblastic response, especially in the leucocyte precursors.

6. Myeloblasts, the non-granular precursors of the myelocytes, were more scanty

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and also shewed no nuclear activity; again in marked contrast to the picture of relapsing fever.

The bone marrow was investigated in this manner in both acute, sub-acute, chronic and complicated malarial cases with strikingly uniform results. For instance, in pure broncho-pneumonic infections the bone marrow consistently showed a leucoblastic response, but in broncho-pneumonia supervening on malignant malaria it was absent altogether, or at the most very slight in degree.

Summary and Conclusions.

1. In severe and inadequately treated malignant malaria the clinical and pathological picture may, even in a short space of time, simulate *very closely* that of idiopathic pernicious anæmia; the cellular picture may be identical, the destruction of the red cells as severe, but the colour index is invariably below normal.

2. In early pernicious cases there is a definite leucopenia both in the pyrexial and apyrexial periods and even with secondary complications there is not the corresponding leucocytic response; it may be altogether absent.

3. These phenomena, as seen in the peripheral blood, are but an external expression of what is happening in the bone marrow. Here red cell production proceeds side by side with elaborate phagocytosis of effete and damaged erythrocytes.

At the same time there is an actual reduction in the precursors of the leucocytes; whether this is due to a direct inhibition by the malarial toxin, or whether the excessive erythroblastic response exhausts the leucogenic activity of the primitive metrocyte, is a problem for future experimental pathology to elucidate.

4. The frequency of secondary infection in malignant malaria, in which a leucopenia is such a characteristic feature, is in direct contrast to a disease like relapsing fever in which leucocytosis is the rule and secondary infection exceptional. (In a series of sixty-two autopsies in this disease, with the exception of five associated parotid infections, secondary complications were entirely absent.)

The sum total of this investigation suggests that it is to the bone marrow and the consequent inhibition of the leucoblastic mechanism we must look for the frequency and fatal nature, often after trivial initial lesions, of these tragic secondary infections in malignant malaria.

To the clinician it obviously bears a lesson to redouble his care in the preparation of quinine for injection and in methods of administration; for sepsis, once introduced from without, obviously finds in such feeble and anæmic subjects the ground well prepared for its rapid spread.

As a result of this, too, the question of transfusion in severe hæmolytic anæmias naturally arises. We would emphasize the necessity for extra care in the selection of a compatible donor by agglutination and hæmolytic tests for the presence of isoagglutinins and hæmolysins, considering the proclivity to hæmolysis in malarial infections.

Acknowledgment.—For clinical assistance during the investigation we are indebted to Lieut.-Col. C. Bickerton Blackburn, O.B.E., R.A.M.C., and to Col. W. Summons, for permission to publish these notes.

The President: Colonel Fairley is at a disadvantage in reading his paper at a very late hour, and I am afraid that, under these circumstances, there will not be much discussion on it. Nevertheless, we thank him very heartily for the careful observations he has made, and, I am sure, it will be a most useful paper for us to study when it has been printed.

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CORRESPONDENCE.

THE EDITOR, TRANSACTIONS OF THE SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

SIR,

In the discussion following Dr. MANSON-BAHR's paper on Bacillary Dysentery, Dr. C. M. WENYON is reported to have said that he wanted to see a large series of cases treated alternately, with and without serum, but that no one was daring enough to risk his reputation in such an experiment. At No. 21 General Hospital, Alexandria, I for a considerable time conducted such an experiment. When I received two acute cases, as similar as might be regarding duration and intensity, the one was treated with serum and the other without. I was never able to persuade myself that the serum cases did better than the others, and, finally, I almost entirely abandoned the use of serum. As my mortality during three summers among cases I had treated from the onset was nil, I do not think it can be contended that my cases suffered.

There were many striking recoveries after serum, but there were equally striking ones without.

I strongly maintain that the usefulness of anti-dysenteric serum is by no means established.

HUGH MILLER, M.B., CH.B.

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Proceedings of a Meeting of the Society held on Friday, May 21st, 1920, at 8.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Professor W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P. (*President*), in the Chair.

THE ETIOLOGY OF PELLAGRA FROM THE STANDPOINT OF A DEFICIENCY DISEASE.

BY EDWARD J. WOOD, S.B., M.D., D.T.M. (ENG.).

In spite of the tremendous amount of work done in the study of the etiology of pellagra, it must be reluctantly admitted that the solution of the problem is not yet accomplished. It is, however, encouraging to note the great interest which has been aroused, and the high type of scientific research which is being brought to bear on its solution. The Great War has stimulated the interest of a number of brilliant investigators, who were given ample opportunity for the study of the disease, especially in Egypt. It would seem highly improbable that the addition to the field of research workers in pellagra of the large number of British investigators whose interest was aroused in Egypt, could result in anything but an early solution of a problem which has vexed Europe since 1735, and which during the past decade and a half has taken such terrific toll in the United States.

My only justification for appearing before this Society to discuss the question of the cause of pellagra lies in the hope that it may help to form a link in the chain of evidence, ultimately to result in a solution, for all my observations were made in the southern states of the United States, and chiefly in the State of North Carolina, where conditions of living and general hygiene are far different from those encountered by the British observers in Egypt. It is reasonable to presume that light may be thrown on the subject by a consideration of its occurrence in areas so widely separated.

I ask your indulgence in a rapid review of some of the more important work done in the United States and the more recent Egyptian reports.

It is interesting to note that there was no reference to the occurrence of pellagra in America until about 1907,[1] though an occasional case was reported of a sporadic nature.

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Careful consideration of the matter convinces me that it is a great error to assume that the disease has existed in that country unrecognised for many years. I saw my first cases in 1905, and took great care to consult the older physicians in the fields of its occurrence to learn if they had ever seen it, but it was as strange to them as it was to me. It is, further, a notable fact that from 1905 to about 1909, the disease assumed a very malignant and fulminating type. Since that time it has gradually assumed the character of the chronic process described in Italian literature. This fact cannot but recall to one's mind the story of the visit to the Fiji Islands from Australia in 1875 of H.M.S. Dido, with measles aboard. According to the account of Sir WILLIAM OSLER, 40,000 out of 150,000 population died in four months. It was presumed that this was virgin soil for measles, and that since that time the death-rate has gradually approached the usual normal. Certainly, a like state of things occurred with pellagra, as I saw it in North Carolina in those earlier years, while to-day it differs little from the Italian type of the disease. This single point has been a continual obstacle in the way of accepting any view which excluded an infectious element.

In all the recent work done in the study of pellagra from a standpoint of a food deficiency, it must be admitted that there is still lacking an entirely satisfactory explanation of the seasonal incidence of the original outbreak as well as the recurrences. Dr. SAMBON[2] has presented some very strong points in favour of a short period of incubation which cannot be overlooked or fail to receive the most serious consideration. His carefully recorded instances of such cases as that of the parents in a pellagrous district suffering from the disease, their removal to a non-pellagrous district where non-pellagrous children were born, and the subsequent return after some years to the pellagrous district which was followed by the appearance of the disease in the children, could only be ignored by the prejudiced.

JOBLING and PETERSON[3] studied pellagra in the city of Nashville, Tennessee, and concluded that there was a connection between the incidence of the disease and poor sewage disposal. Their conclusions suggest that a lack of screening and the part played by flies could not be ignored, though they emphasised the fact that the pellagrous class consumed relatively more carbohydrate and less protein than is required to make up a properly balanced ration.

The THOMPSON-MCFADDEN[4] Pellagra Commission made its observations in Spartanburg County, South Carolina. They concluded the disease was in some way related to a bacteriological infection, and they, too, regarded the disposal of human excreta as playing an important part.

The work of JOSEPH GOLDBERGER,[5] with which you are doubtless familiar, has for its basis (1) that pellagra is essentially of dietetic origin; (2) that it depends on some undetermined error in a diet with a disproportionately high vegetable component and a disproportionately low protein component derived from animal or leguminous sources; (3) that no pellagra developed in those who consumed a mixed, well-balanced diet which is suitably varied. In an orphanage, GOLDBERGER found 75 per cent. of the children pellagrins in 1913, and the ones who escaped were the younger children whose diets were supplemented with cow's milk. His suggestions of a diet reform were adopted, and in 1914, in the same institution, among 234 inmates there did not occur a single case. He then selected what his experimental work indicated to be a faulty diet, and by an arrangement this diet was given to prisoners. Every effort was made to exclude all possible sources of error based on the various theories of etiology. A careful survey indicated that the only source of hygienic error rested in the diet. Resulting from this feeding experiment, a number of the men developed a disease condition regarded by GOLDBERGER and clinicians who had seen much of the disease as real pellagra. It is only fair to state at this point that there was criticism raised by some of the opponents of a food origin of the disease, because the skin lesions of the subjects of the experiment were on the scrotum rather than the exposed portions of the skin surface. This led me to review my cases of pellagra, and I shall shew you photographs which readily sustain the point that the covered portions of the body surface may be the seat of the lesion. In a number of cases I have found the skin lesions about the vulva, on the back, above and below the knees,

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behind the knees, and, in fact, hardly a spot on the skin surface can be regarded as exempt from such a possibility. Too little attention has been paid to these atypical distributions, and it would be exceedingly unfair to discredit GOLDBERGER's important work because of a lack of such knowledge.

GOLDBERGER then fed by mouth the fæces of pellagrins to a number of volunteers. In no case did anything suggestive of the disease appear as a result.

It is an important fact that in the United States the instances are neither single nor isolated where a dietetic reform in public institutions has resulted in a disappearance of pellagra, just as GOLDBERGER demonstrated.

W. H. WILSON,[6] in Cairo, has contributed importantly to the subject. He regards pellagra as due to a defective protein supply in the diet. His work resulted from careful analyses of diets known to have produced pellagra, and with others regarded as preventive or curative. He it was who directed attention to the biologic value of protein in such cases. The work of K. THOMAS[7] was depended on for the values of protein from various sources. To maintain nitrogenous equilibrium it was found that various substances differed widely. The biological value of vegetable protein, and especially that derived from maize, was found to be less than that of milk or meat protein. THOMAS estimated that the amount of tissue repair from 30 grams of meat or milk protein required 100 grams of maize protein to be equalised. It was thought that the inferiority of maize proteins could be explained by the large proportion of zein, which is devoid of both tryptophan and lysine, two amino-acids regarded as essential for animal nutrition. WILSON concluded that the danger from pellagra occurred when the biological value of the protein of the diet was below a certain point.

F. B. BOYD[8] and his co-workers studied pellagra in Egypt among prisoners of war. They found that the level of biological protein could be so exactly determined that they were able to anticipate the disease. Their work pointed strongly to the fact that a rest diet was inadequate to protect against pellagra when the victim was put to work. In their series 2,000 German prisoners, who supplemented the reasonably good prison diet in various ways, according to the well-known German appetite, escaped. But among 6,000 Ottomans, not so fortunate as to be able to have this supplement, 300 cases of pellagra developed in one year. It was found that if the disease was not far advanced it promptly responded to an increase in the biological protein value. These workers emphasised the importance of the part played by diarrhœal disease in disturbing the protein balance.

A recent summary of this Egyptian work shews that the absorption of protein from the intestines was less than 67 per cent., and that the metabolism of the Turkish prisoners of war is characterised by a low degree of assimilation of fat and nitrogen. At this point the justification of this communication seems apparent, for in my own work in America, on a carefully weighed Schmidt-Strassburger test diet, a normal utilisation of both fat and nitrogen occurred in pellagra, even in the terminal stages of a fatal case.

It has always, even since the days of CASAL, been a noteworthy fact that those living in the country districts were the victims of pellagra, while the urban population escaped. The Egyptian workers have proven, by careful analysis, that rural diets have lower values than urban diets, as there is a tendency for the agriculturist to send all his expensive animal foods to the better paid town dwellers and live chiefly on cereals. They suggested that the hill-man, depending more on his flocks for food, was more protected from pellagra than the valley-dweller, who largely depended on his cereal crop for his own sustenance.

A. D. BIGLAND,[9] in an admirable review of the whole subject, refers to the biological protein value of WILSON, as indicating the greater readiness with which meat protein is assimilated than the vegetable type. It is this assimilability which constitutes its biological value, and this point is placed by WILSON at 40 grams per diem. BIGLAND concludes that pellagra occurs most often in the underfed, speaking exactly, but may occur in the well fed. He speaks of the food deficiency being from without or from within. A comparison is made with perilobular cirrhosis of the liver, which may occur in the alcoholic but also in the non-alcoholic. This broader point of view may make possible an explanation of many of those baffling cases in which we have seen pellagra occur in the well-fed.

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J. I. ENWRIGHT[10] reports on the occurrence of pellagra among German prisoners of war, who were given the very "fat of the land" in the way of food, and whose dietary, which is published in detail, will stand the closest scrutiny. In spite of such a diet definite pellagra occurred.

CHITTENDEN and UNDERHILL,[11] in the United States, produced a condition in dogs very suggestive of pellagra, on a diet which E. V. MCCOLLUM[12] found defective only in Fat-soluble A, and the latter's investigations on rats with the same type of diet resulted in failure to produce the pellagrous symptoms, but did bring about a retardation of nutrition and a condition approaching xerophthalmia. MCCOLLUM thinks there is good evidence that there occur sometimes cases of pellagra in individuals whose diets have included a certain amount of those things called by him protective foods, which include milk, eggs and leafy vegetables, and that the theory of infection is thereby supported. He thinks this view is supported by the occurrence of the disease in badly sewered districts.

Working under the suggestion of GOLDBERGER, after twelve years of a belief in an infectious nature of pellagra, I undertook to view it from the standpoint of beriberi. I was at once attracted by the report of P. A. NIGHTINGALE,[13] who had under his care the prisoners of a gaol in Rhodesia. These prisoners had always been fed on ropoko, a small cereal of the maize type, which was hand-ground by the prisoners, but owing to the failure of the crop they were forced to go outside and buy commercial "mealie meal," which seems to conform to the commercial corn meal of the Southern States of America. The result of this food change was a disease the description of which is accompanied by diagrammatic sketches, making the diagnosis unmistakable. Appreciating that the trouble was due to the change of food, NIGHTINGALE immediately abandoned the "mealie meal" for ropoko again, and, in his own language, the result was "immediate and magical." It is to be hoped that my hearers will appreciate the difference between this observation and the old maize theory of the early Italian School. In NIGHTINGALE'S observations there was no thought of toxicity but rather of a deficiency.

If pellagra is a deficiency disease, it may occur as often among those who eat wheat as among those who eat maize as the exclusive cereal diet. In either instance it is safe to say that milk, or other suitable animal protein, may readily offset the fault. So much impressed was I with NIGHTINGALE'S report, that I went to the maize-mill to learn how the grain suffered in the milling process. Briefly I[14] found that in modern steam-milling the grain was "degerminated," that is, it was deprived of the germ, which latter was ground separately, and, with the bran, sold as cattle food, having a great reputation as a "milk maker." It will be recalled that the Philippine workers, in the experiments with polished rice, rice-polish and beriberi shewed that phosphoric acid (P_2O_5) was a reliable indicator of antineuritic substance. In my maize work the same plan was adopted. We found that the commercial germ product contained 1·15 per cent. of P_2O_5, while specially milled germ for experimental purposes often reached as high as 2 per cent. On the other hand, maize milled in the state of Ohio contained only 0·29 per cent. When the grain was ground *in toto* with no removal, according to the old plan, the P_2O_5 content was 0.78 per cent. The reason for the modern plan of milling is that the germ of maize contains so much fat that milling the grain without separating and removing this germ results in a product which early becomes rancid. The germ of maize is quite soft, and is poorly protected. If deterioration of an animal or vegetable source occurs, it is always the germ which suffers first and foremost. Is it not worthy of consideration that, if LOMBROSO had regarded pellagra as a deficiency rather than a toxicity, resulting from the destruction by the various moulds of the essential portion of the grain, a solution might have been reached? Substituting the word deficiency where he speaks of toxicity, makes his masterly work on the subject in close accord with many modern views. I then investigated the milling of wheat. It will be recalled that the antineuritc substance occurs in the cortical portion of the grain. Our analyses shewed that the bran portions of wheat contained 0·98 per cent. of P_2O_5, while average wheat contained only 0·14 per cent.

Experiments were undertaken on pigeons and chickens, having no point of originality except the substitution of deficient maize in place of polished rice in the production of

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polyneuritis gallinarum. It was readily proven that the germ of maize contained the same property as the polish of rice. An alcoholic extract of the germ of maize was as effective in relieving the symptoms of polyneuritis as was the case with an alcoholic extract of rice polish. In the course of these experiments it developed that birds given grain, the phosphoric acid content of which was very gradually decreased, seemed to acquire a tolerance for the deficiency. But the young of these birds hatched from eggs laid during the experiment, while apparently free from polyneuritis at the hatching time, and continuing so until after leaving the nest, promptly became polyneuritic on the diet of the parent birds, though these latter remained free from any definite indication of the disease. Pigeons, on a diet of maize with a phosphoric acid content considerably below normal and yet not low enough to produce polyneuritis gallinarum, manifested droopiness, loss of feathers, and a red-legged condition which seemed quite distinctive. We have not yet dared to say that this condition is the analogue of pellagra as polyneuritis gallinarum is of beriberi. Pigeons fed on whole maize of the best quality were maintained in fair health, but without rapidity and extent of growth which would occur on a more natural diet. The condition of red legs was readily relieved by a substitution of good maize.

Repeating what has been done in the rice work in the Philippines by VEDDER and others, it was found that cooking the grain with sodium bicarbonate added materially to its deficiency.

I have cured polyneuritis gallinarum in the fowl by the administration of fresh cow's milk in almost as striking a manner as with the alcoholic extract of the antineuritic portion of the cereals.

Maize is frequently the bread cereal in pellagrous countries. The same experiments detailed above have been done with wheat. It is not then a question so much of the particular cereal as it is of the portion removed in the milling.

It would appear that the deficient portion of the several cereals in these experiments was the water-soluble B. The part played by milk, in my experiments in curing polyneuritis gallinarum, may have been due to the small fraction of water-soluble B, though it remains to be seen whether or not in pellagra the dietetic fault lies in the lack of one or both of these elements. The following experience illustrates the point. Applying the principle underlying NIGHTINGALE'S work, together with the suggestions derived from animal experimentation, I fed pellagrins with the germ of maize with notably prompt improvement. In one particular case the patient was *in extremis*, and was being fed by the tube. The food given in this way was eight ounces of fresh cow's milk, three eggs and one or two ounces of sugar of milk. There was no notable improvement until a gruel, made of wheat bran, was added, when at once the general condition of the patient greatly improved, though she eventually died the sudden death which I have so frequently noticed in this disease.

It is obvious that the pellagra class as a rule could never secure the expensive protein which would protect, regardless of the type of deficiency underlying the disease. Since this economic solution does not lie within our reach, it becomes the more important exactly to determine the type of dietetic fault, in order that it may be practically corrected with as little expense as possible.

Our experiments led us to suspect that the water-soluble B was more at fault by its lack than the more expensive fat-soluble A. Applying this suspicion practically, we have advocated the consumption of the whole cereal without the removal of a part in the milling and the cooking of the cereal without bicarbonate of soda as a rising agent. We found that the balance between acid and alkali in many baking powders was so crudely struck that for the present, at least, we are advocating that they be avoided. The results are already encouraging. Whenever possible, milk, fresh meat, vegetables and other sources of fat-soluble A are added.

If the problem is one simply of a too low protein level, or a too low biologic protein, there are many classes of sufferers that cannot be reached. If, however, we can finally determine that water-soluble B is the deficient element, it can be readily supplied at small cost. There is still much more work to be done before this matter is settled.

Many were impressed during the war with a belief that while beriberi may be a

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deficiency disease, there must have been added an infectious element. One strong point along this line mentioned to me by a careful observer was the occurrence of jaundice. Surely, if this is a reasonable suspicion in the case of beriberi, the case of pellagra cannot be counted solved, at this time, especially in the light of such observations as those of ENRIGHT, JOBLING, the THOMPSON-MCFADDEN Pellagra Commission, MCCOLLUM, and finally, but probably most important of all, the epidemiological observations of SAMBON in Italy, Roumania, the United States and the West Indies. That nutritional errors do play a large part I do not doubt, nor do I believe that an individual on a well-balanced diet, especially when milk plays a part, will develop the disease, but I am unwilling at this time to exclude the possibility of an infectious element playing a part which may be secondary but no less essential.

In North Carolina, it was noticed that pellagra occurred chiefly in the foot-hills below the high mountains; that, as a rule, along the Atlantic seaboard the incidence of the disease was much less. Our cotton-mills are located in the foot-hills of the mountains, chiefly because of the water power so easily had there. In the high mountains the disease did not occur except very rarely. At one time it looked as though this distribution would coincide with the distribution of some insect pest, but such has not been demonstrated. There appeared to be a direct relationship between the lines of railroad travel and the occurrence of the disease. One might view this as an indication of an infection. On the other hand, I have observed that there seems to be a connection between the appearance of the disease and the introduction of such manufactured products as commercial maize meal and patent wheat flour, supplanting the old primitive plan of taking to the local water mill a small portion of maize, which was ground *in toto* and consumed before rancidity could occur. So far as a careful investigation of the manner of life of the people far removed from railroad travel and those living on the line of the railroad goes, the only real difference consisted in the manner of milling and cooking the cereal. The mountaineer in many instances violated every hygienic rule, and his diet was certainly not ideal, but he escaped the disease. On the other hand, the dweller in the foot-hills, working with all his family in a cotton mill, took whatever the local merchant provided and adopted all measures of shortening and making easy the cooking process. Consequently, the latter got wheat flour with no cortex, maize meal milled without bran or germ, and all manner of chemical rising agents. With this latter class, pellagra finds its greatest number of victims. It was interesting to trace the outbreak of pellagra in a village, timing its appearance with the abandonment of the old grist mill to which each farmer carried his small portion of cereal for primitive grinding. There seemed to be a striking coincidence in the appearance of the disease with the adoption of modern errors in milling and cooking.

The American pellagra problem differs from the Egyptian, essentially in the fact that diarrhœal diseases are relatively infrequent in the pellagra class. It is true that those infected with the *Necator americanus* fall an easier prey, and that we have much more amœbic dysentery than is usually thought, but, in spite of these exceptions, it must be granted that the element of chronic diarrhœa, so much stressed by the Egyptian workers, will not materially befog the atmosphere, and that the application of the Egyptian findings to our problem will be most helpful and instructive.

While the time has not arrived for a definite conclusion, we have found in North Carolina that, for all practical purposes, a careful analysis of each diet and a correction of its errors bring protection, and oftentimes relief.

I acknowledge with appreciation my debt to Captain GEO. F. CATLETT, of the United States Army, for great aid in the chemical portion of this work.

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DISCUSSION.

The PRESIDENT: Dr. WOOD, with his great experience of pellagra, has given us a very valuable and suggestive paper. He has examined the different views held in regard to the causation of the disease, and he has done it in a modest and scientific spirit. He has given credit to those who have worked seriously at the subject, while he has upheld his own views in a very convincing manner. This attitude is very different from that of a recent writer, who, I am afraid, does not quite comprehend the scope of the enquiry into pellagra instituted by the British Committee, and parodies the conclusions without apparently taking the trouble to study the reports connected with the investigation. Dr. WOOD, on the other hand, has made very important observations, and he shews, as many other investigators have, that the incidence of pellagra falls particularly upon those whose diet is at fault. He has shewn clearly, on the map and in the paper, that the incidence of pellagra in the country in which he has worked has fallen chiefly on the inhabitants who live on the foot-hills, and whose diet consists largely of wheat flour without the cortex, and maize that is milled and loses its germ. He also attaches very great importance—and has good reasons for it—to the fact that in using various chemicals for baking purposes, for the raising of the bread, the last straw is put upon the vitamines which may have been left. He differs from many observers in thinking that the water-soluble B is more important than the fat-soluble A; and certainly, if his views are correct, great economy will be practicable, whereas in the "no-protein" view there is likely to be a difficulty in practice owing to expense.

With regard to these nutritional diseases, we find that they are increasing largely in number. We now have, according to some, leprosy added, and tubercle as well We were always under the impression that these people were fed badly, but now we seem to be getting to the root of the matter as to the particular element which is wanting. But if leprosy and tubercle are deficiency diseases, they also have with them an infective element, and I am glad to see that Dr. WOOD does not lose sight of the fact of the extraordinary virulence of pellagra in its first invasion of his country. The sudden spread of the disease over a large part of the country is a matter which must not be lost sight of, and supports the view that there is some other agent associated with the deficiency element.

I take it this paper shews that the solution of the problem has not yet come—that it is necessary for further research to be carried out.

Dr. P. MANSON-BAHR: In the absence of Drs. BIGLAND and ENRIGHT, I have been asked to relate some of their extensive experiences of pellagra in Egypt, and to shew

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some of the paintings of pellagra cases amongst German prisoners. I had the advantage of being associated with much of this work during my residence in Egypt. At first there was much to be said for the food-deficiency theory. The first pellagra patients we got in Egypt were Turkish prisoners from Arabia. Those who have followed the literature in the *Lancet* will have observed that, although some of us had our eyes open to the possibility of the occurrence of pellagra, we were at first completely mystified. In the first prisoners who came under observation, the only objective symptom was enormous œdema, which afterwards Dr. BIGLAND looked upon as analogous to the war œdema which was occurring in Europe. In January and February, 1916, some of these people commenced to have a typical pellagra rash. The evidence went to shew that, in some cases, these were recurrences of a long-standing disease, and that the œdema was an epiphenomenon. Most of the Turkish prisoners were affected with intestinal disease of some sort or other, and post-mortem bacillary dysentery lesions were common, though amœbic dysentery seemed to be rare. The low protein value of the diet and the scrappy existence these people had led in the last year or two seemed to favour the food-deficiency theory. In 1916 there was a serious outbreak amongst the Armenian refugees at Port Said, and that also could be explained on a food-deficiency basis. From the middle of 1917 onwards I had not the opportunity of following this subject further until the beginning of 1919, when, just after the Pellagra Commission had finished its work, out of the 7,000 Germans taken prisoner in Palestine the previous autumn, a considerable number started to develop typical pellagra, as you will see from the drawings. From January to June, 1919, when the observations ceased, there were sixty-five cases, that is, the disease occurred in approximately one per cent. of the German prisoners. Some were very ill, and, I think, one died. Nothing could be more typical than the distribution of the rash, even including the anomalous patches on the elbows, the inner side of the knees and the popliteal space. And, as far as one could make out, there was no reason to suspect that these men had ever suffered from any obvious food shortage. One outstanding fact was that two of the men were prisoners from German East Africa, who had been in prison camp at Maadi for two years—the very camp where the diet was favourably reported upon. Therefore, it seems we were up against a paradox. It may be said that the biological value of the protein was affected by the amount of the intestinal disease these men suffered from. But it will be seen that this is not the determining factor; they were not afflicted with malaria or dysentery more than our own men, and some of them were in the best possible condition, and on capture they looked remarkably healthy. One was a gymnastic instructor in the German army, and only had two months in Palestine previous to being captured.

The main difficulty to us was how to account for this outbreak of pellagra amongst apparently healthy Germans, who said they lived on "the fat of the land" while in Palestine—meat, fruit, milk and eggs, and who, a short period after their captivity, developed this disease, especially when they were thrown into close association with the Turks. That seemed to favour the idea of pellagra being an infectious disease. Considering the problem as a whole, how is it that, with all the food deficiency that existed in Central Europe during the war such as the distressing conditions in Austria, no epidemic of pellagra has been reported? And how is it that during the whole of the war, when our men have been in close association with natives who were known to be stricken with this disease—for instance, with the Egyptian Labour Corps, a proportion of whom were pellagrous—not a single case of pellagra has been reported as occurring among British troops, or amongst the hospital personnel who attended pellagrous Turks over a period of three years?

It seems to me that we have reached a critical stage in the investigation of pellagra. Perhaps too much is being made at the present time of deficiency in diets in general. Sprue and pellagra have many features in common. No one who has seen much pellagra and sprue can help being struck by the similar appearance of the mucous membrane and the tongue in the later stages, especially the similarity of the stools. From chemical and biological investigations it seems that the onus in those two conditions falls on the endocrine glands: in sprue, on the pancreas and the organs of digestion; while in

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pellagra it would seem to be the suprarenals and the sympathetic nervous system in connection with those glands which bear the brunt of the process. There is also much similarity between Addison's disease and pellagra, and it seems that the branch of research which will bear more fruit at present than anything else is one which directs attention on the suprarenal gland. Is there some toxin acting specially on these glands, and is that toxin peculiar to the countries where pellagra occurs?

Lieut.-Colonel S. P. JAMES: I notice that although Dr. WOOD has added a lot of valuable information in support of the view that pellagra is a disease resulting from a faulty diet, he has not satisfied himself that it is the sole explanation. And he has told us that the food-deficiency theory does not account for the marked seasonal incidence which is the most striking feature in the epidemiology of the disease. I do not think anyone has been able to shew that in pellagrous districts the food varies materially with the seasons of the year, but, unless there are these variations, the faulty diet view is not a satisfying explanation from the epidemiological standpoint.

Dr. WOOD says there may be an "infective element," secondary to the food deficiency. If we admit that there must be two factors simultaneously at work to cause pellagra, is it necessary to say one of them is of an infective nature? Perhaps we are apt to dismiss too lightly the old idea of the effect of sunlight in causation. Dr. WOOD knows quite well that if one takes the statistics of South Carolina and plots them as carefully as one can, and then takes the hours of sunshine and plots them out, the apparent relationship between the two is remarkable. The peak of the maximum sunshine is about a month before the incidence of pellagra. A similar relationship is apparent between sunshine data and pellagra incidence in Massachusetts and other States.

Lieut.-Colonel P. S. LELEAN: I have listened with very great interest to Dr. WOOD's paper, and also to Dr. MANSON-BAHR's remarks. As I had the privilege of working with Colonel BOYD on the Egyptian Committee of Enquiry into Pellagra, I think it would be unfair to him if I were to fail to point out certain facts, particularly in regard to the German prisoners.

With regard to the alternative theories of infectivity and food deficiency, it is interesting to consider the source of pellagra among Turkish prisoners of war. On very close investigation into the history of 440 cases among such prisoners—enquiries carried out by their own medical officers—it was found that only one of those patients had seen in his native home a corresponding rash, and only five had seen a similar rash among his acquaintances. Morever, thirty medical officers among the Turkish prisoners of war all denied that the disease existed in Turkey. Therefore, the occurrence of pellagra in the regions from which these men came was negligible. The first four cases of pellagra which could be traced among these prisoners had their initial attacks in Gallipoli. The troops were taken from there and passed to Syria, where similarly only a few cases (21) occurred—obviously among the very susceptible. What was the source of infection in Syria, if infection it was? Amongst the population there, medical officers who had had considerable experience in general practice stated that it was comparatively unknown, though they were acquainted with the description of it in the text-books. One had been fifteen years a practitioner in Damascus, but had seen only one case among the population there. When the Turkish troops came down into Palestine, pellagra came on in proportion as lengthened and overtaxed lines of communication led to failure in the supply of adequate rations. In captured documents we found evidence that their food was reduced to 67 per cent. of the authorised ration, and that it provided 24 per cent. less protein than is required for the performance of light labour, though they were engaged in arduous entrenching. Of these 440 cases which were closely investigated, the tabulated results shewing the onset of initial symptoms bore the following relation to the date of capture of these prisoners:—

12 to 6	months	before	capture	=	40	cases.
6 „ 0	„	„	„	=	279	„
0 „ 5	„	after	„	=	27	„
6 „ 12	„	„	„	=	13	„

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If an infective origin is argued, how do you account for these figures, and for the fact that the men did not carry the power of infection into captivity? And why was the fall from 279 to 27 so sharply defined by the period of capture, *i.e.*, the passing on to a better diet? Remembering the indefiniteness of the initial symptoms, most of the dates of first onset must be regarded as recurrences.

We may further consider the evidence as regards infectivity which is afforded by an outbreak of pellagra in the Armenian refugees' camp on the Suez Canal. They were maintained in the same camp, with no change of site, and no altered range as regards biting-flies. There is no doubt that these people were subject to no materially altered condition, other than those of diet. After four months' exposure to a low diet, in which the biological value of protein varied from 22 to 27 grammes a day (adult male equivalent) they began to get pellagra in marked degree. Then the men went to work and got money, which they sent home, more food was purchased, and the pellagra rate fell. Then the men struck work and returned; they consumed the diet which otherwise their families would have found adequate, and up went the pellagra rate. The biological value of the diet in protein was raised above 40 grammes per diem (on the adult male standard), and coincidently with this increase the pellagra prevalence fell until it ultimately reached zero. There was subsequently no recurrence, although the people were living under the same conditions, except during a short period when there was a temporary diminution in their rations.

With regard to the German prisoners there is some difference of opinion. There was probably nobody in Egypt who had a more highly trained acumen for early cases of pellagra than Colonel F. D. Boyd. He had followed up many cases from the earliest diagnosis, and was able to detect pellagrins a month earlier in their development than were untrained or partly trained medical officers. He went down and saw those prisoners at Maadi Camp. Some had been there for over two years—since one of the enemy sea raiders had been run ashore in East Africa. They had not had a single case of pellagra during that year. Colonel Boyd examined 200 of these men, who were taken haphazard, and there was only one at whom it was necessary to take a second glance. The evidence is thus conclusive that at that time pellagra did not exist among those 2,000 German prisoners, although the 6,000 Turks in an immediately adjoining camp, and under precisely the same general conditions, were getting pellagra at the rate of 300 a year. We have just heard of pellagrous German prisoners "living on the fat of the land." Their authorised ration contained 54 grammes biological value of protein, 28·8 grammes of fat, and 351 grammes of carbohydrates, giving a total calorie value of 2,079—if this be "the fat of the land," the inhabitants thereof are not very gross feeders. As has been pointed out, the ratio of biological protein to the dietetic needs of the individual depends primarily on the extent to which it is assimilated, and, secondly, to its reaching the tissues. Here we had men who were only getting 2,000 calories, and the otherwise ample biological protein would not have reached their tissues, but would have been "deviated" to meet the immediate energy needs of the body, unless these had been met by augmentation of their ration by purchased food. The Germans were allowed two shillings a day, and, at the time of Colonel Boyd's inspection, they were augmenting their diet by practically the whole food value of that sum, so that their calories mounted up to 3,590. The essential point which must be considered is whether these Germans were at a later stage still receiving this money as Germany came nearer to bankruptcy. If not, they may have developed pellagra—as we should have expected—but they would certainly not have been living on "the fat of the land" at 2,000 calories a day; but, it must be remembered, that long-continued individual failures in assimilability may, in rare instances, determine an attack of pellagra by starving the tissues of protein, in spite of a most generous diet.

I owe it to Colonel Boyd that I should make these remarks, for he went into the investigation of these German prisoners with extreme care.

Dr. W. H. Willcox: The special point which interested me was Dr. Wood's attempt to supply a theory which would explain pellagra. I have not had the opportunity of seeing much pellagra, but the theories Dr. Wood put forward are exactly those which

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have been advanced to account for beriberi. It was noted, in the cases which occurred in the Dardanelles and in Mesopotamia, that in addition to the deficiency in the diet, the water-soluble B element, there was an infective element. And in a paper I published, in 1916, on the relationship of jaundice to beriberi in the Dardanelles cases, this was pointed out. I would like to ask Dr. WOOD to explain why, according to his theory, pellagra should develop, when, under the same conditions, viz., deficiency of water-soluble B element and an infective element, beriberi is well known to be caused.

If Dr. WOOD's explanation of the cause of pellagra were correct, surely an outbreak of the disease will be associated with a number of cases of beriberi. There does not appear, however, to be any evidence of association of outbreaks of the two diseases, so that there must be a difference in heir etiology.

Mr. T. P. BEDDOES: The great merit of the paper we have just heard, is that it presents such an admirable epitome of the various facts bearing on pellagra. What struck me most were the figures concerning the skin lesions. As far as one can see, it is possible the variation in the skin lesion depends on the degree of exposure to heat The lesions on the inner side of the knees may occur in those who live indoors and are in the habit of sitting facing the fire, with toes turned outwards, so that the affected part of the knee is that which comes quite near to the fire. That is the part which is frequently affected with chronic eczema, which may become so chronic that the skin is thickened, and it is then known as lichenization.

With regard to the cases of Dr. GOLDBERGER, I ask whether my recollection of that paper is correct, that those men were kept in a separate building which was well shaded from direct sunshine.

As to seasonal incidence, we must remember that when pellagra was first recognised, the first characteristic lesions were skin lesions, and if they were absent, some hesitated to regard the case as pellagra at all. The next lesion was the intestinal one, producing disorders of digestion, and the third was the mental. The seasonal incidence which was reported depended on which of those disturbances was recognised as characteristic. A large number of advanced pellagra cases were found in lunatic asylums. Dr. MANSON-BAHR has shewn that pellagra is now recognised at a much earlier stage than it used to be. One would like to know to what extent the lesions which occur on covered parts of the body are due to people living in the shade. Egypt, of all countries, is an unwooded country, whereas America is, perhaps, the most wooded country in the world.

With regard to the view that the sudden appearance and spread of pellagra in the southern states of America is in favour of infection, my recollection is that statistics in the *United States Health Bulletin* shew that the appearance of pellagra coincided with the spread of industrialism, causing decreased food production, decreased food values in the dietary from influx of population into the towns, in fact, that the rapid increase of pellagra is in favour of dietary deficiency and against the infection theory.

Dr. P. MANSON-BAHR: I would like to assure Colonel LELEAN I did not wish to criticize the Pellagra Commission report in any way. My only difficulty is that I cannot fit in all the recent facts with their conclusions. When I spoke of living on the fat of the land, I referred to the German prisoners who were taken in our last advance, not those who had been living in prison 13 months or more. The official German Army ration in Palestine which was given to Dr. ENRIGHT by the sergeant-major in charge of the ration dump at Damascus, was a very generous one: 750 grammes of bread per day, 453 of fresh vegetables, in addition to beans, rice, potatoes, and 350 grammes of fresh meat and 30 grammes of sausage, and sugar and coffee, milk and eggs. A striking fact was that one man developed pellagra after he had been only two months in Palestine before capture. He was five months in captivity before he developed a pellagrous rash, and quite a number of Dr. ENRIGHT's cases became pellagrous whilst in hospital suffering from other diseases, such as malaria or dysentery.

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Lieut.-Col. P. S. LELEAN: In reply to Dr. MANSON-BAHR, the figures I gave were those of the official ration, and there is no reason to suppose they are inaccurate. It is the scale laid down by G.R.O I understood Dr. MANSON-BAHR to say that on the average prisoners had been in captivity three months. Our observations indicated that a four months' dietetic shortage preceded the outbreak of pellagra among the Armenian refugees. The case he has just quoted of the prisoner who had been in captivity five months, just brings him into the period at which one would expect pellagra to break out under a dietetic shortage, but one has to remember that the vast majority of cases reported as "fresh cases" of pellagra among prisoners of war in Egypt, proved on close enquiry to be recurrences. In 1919, the D.M.S., E.E.F., reported that of the "primary cases" investigated in that year, "Practically everyone has had previous symptoms and hospital treatment for the disease."

Dr. E J. WOOD: I have presented the case to you as I found it, without reservation. In some things I was able to form an opinion, while in many points the facts were left for discussion in the hope that light might be thrown on the difficult places I have received such a wealth of help in this study in London, that I hoped to be able to contribute to the work a small link in the chain from my American experience.

The idea of an infection is not mine, but it seems inexpedient to reject it until we have more conclusive proof. The Thompson-McFadden Pellagra Commission had the advantage over the Egyptian Commission, in that they were able to follow their cases through several years, and their conclusion is that the disease is an infection controlled by the proper disposal of sewage. Added to their work, is what we hear now from Major MANSON-BAHR, who corroborates ENRIGHT in the suspicion of an infection. In the idea of infection McCOLLUM, too, is in accord.

Several years ago, Dr. JOSEPH GOLDBERGER came into the southern states teaching the doctrine of an insufficient protein diet. I had for several years represented the North Carolina State Board of Health, in this matter, and was convinced that the disease was an infection. Opportunity soon came to me to go over in detail with Dr. GOLDBERGER a long series of my own cases. At the end of this conference we agreed that I should view my next hundred cases from the beriberi viewpoint. This I did, and on leaving America I regarded the problem as solved, though not agreeing altogether on the nature of the deficiency. The admirable work of Professor BOYD and his associates in Egypt seems to me to be entirely in accord with the work of GOLDBERGER in America. Certainly their conclusions are identical.

In experimenting with pigeons and chickens I found that a diet deficient in water-soluble B, but not entirely lacking in it, resulted in a condition characterised by droopiness, loss of feathers and red legs. I am not prepared to say this was the analogue of pellagra, as polyneuritis is of beriberi. The beriberi nations are usually one-cereal people, and if that cereal is deficient, as in the case of polished rice, beriberi results. My own idea is that pellagra is the result of the same sort of deficiency, but less of it, because the pellagra people are not one-cereal consumers, but always supplemeut the grain with some other food.

In America, many of us regard the problem as solved, so far as prevention is concerned. Wherever GOLDBERGER'S views have been fairly applied, the disease has disappeared. It remains a question for solution, how can this be most practically accomplished? The famine stricken nations can hardly be expected to secure sufficient protein food. If, however, it can be proven that the less expensive water-soluble B is the vital factor, the problem is greatly simplified, for this can be supplied at small cost.

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LABORATORY MEETING.

A Laboratory Meeting was held on 19th March, 1920, at the Royal Army Medical College, when the following microscopic preparations or apparatus were on view or demonstrated :—

1. "Rabies," by Sir STEWART STOCKMAN.
2. "The Amœbæ of Man," by Mr. CLIFFORD DOBELL.
3. "Some of the Pathogenic Fungi," by Colonel S. L. CUMMINS and Major J. C. PERRY.
4. "Development of Malarial Parasites in Mosquitoes," by Dr. C. M. WENYON.
5. "Trypanosomes outside Blood-vessels," by Dr. A. C. STEVENSON.
6. "Scurvy in Animals," by Surgeon-Capt. P. W. BASSETT-SMITH.
7. "The Agglutinometer," by Dr. GARROW.

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REVIEW.

WAR AGAINST TROPICAL DISEASE.

We have received a copy of Dr. BALFOUR's new book, entitled *War against Tropical Disease.* A sub-title explains that it is a series of seven sanitary sermons addressed to all interested in tropical medicine and hygiene. All but one of these, "The Palm from a Sanitary Standpoint," have already been delivered as lectures, and have appeared in print. They have now been revised and brought to date.

It was a happy thought which led the author to collect his sermons in this beautiful volume It is written in his well-known interesting style, and without sacrifice of scientific accuracy the matter is presented clearly, and in such simple language that it is a pleasure to read the book. It cannot fail to appeal, not only to the sanitary expert, but also to the layman who desires to understand something of the fascinating problem of the War against Tropical Disease.

The first Sermon deals with the functions and duties of the Sanitary Inspector, that intermediary between the Medical Officer of Health and the native subordinates. The author is, of course, speaking of his experiences when Medical Officer of Health in Khartoum, where he found that without the British Sanitary Inspector it was impossible to carry out or enforce the regulations which were necessary. He describes the difficulties that had to be overcome, and the necessity of never-ceasing vigilance and labour. Even in most trying circumstances humour is not lacking, and we are told that, on one occasion, the Medical Officer of Health had to fine himself for the discovery by a Sanitary Inspector of a mosquito breeding-place in his own premises.

Sermon No. 2 is an account of the author's journey in the New World, where he visited Barbados, Grenada, Trinidad, Venezuela, Curaçao, Maracaibo, Colombia, Jamaica, Cuba and other places. As the author remarks, it is well on such occasions to be blessed with the sanitary eye, though the sanitary nose may not always be a pleasant possession. It is evident he had the former, from the fund of information presented to the reader. He describes the condition of the "poor whites" in Grenada, the descendants of the original white settlers who came from Barbados. They are ridden with malaria and ankylostomiasis, and he points to this as an opportunity for some wealthy philanthropist to save the offspring of these poor people. The author still wonders if the red howler monkey may not sometimes play a part in the spread of yellow fever.

Sermon No. 3 deals with preventive inoculation against typhoid and cholera, and is a lecture delivered under the auspices of the Research Defence Society early in the war. It presents, in an ingenious manner, the main features of protection of the human organism against bacteria.

The fourth Sermon is entomological, and deals with the noxious insects of Macedonia. It was given as a lecture there during the war, and did much to popularise the bearing of entomology on tropical medicine. Medical officers who served with that Force never forgot the lessons learned on this occasion.

Sanitary and Insanitary Makeshifts in the Eastern War Areas is the title of the fifth Sermon. It shews the wonderful ingenuity of officers in the field, who contrived to meet the sanitary requirements often when practically no material was available. The petrol-tins receive their deserved praise, and the author remarks that he cannot say where we should have been without these aids to hygiene. The persistence with which certain officers demanded material for sanitary purposes, is illustrated by the commanding officer

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who could obtain no wood till he indented for a load of timber to erect crosses over graves. Wood there was for the dead but not for the living. A feature of this sermon is the numerous illustrations of sanitary contrivances.

The sixth Sermon deals with the problem of Hygiene in Egypt, the collected Chadwick Lectures delivered in 1919. The author was President of the Commission to enquire into the working and organisation of the Public Health Department of Egypt, and no one was better qualified to speak on this subject. The lectures review the whole of the sanitary conditions of Egypt, and indicate the lines along which improvements should be made.

The final Sermon, on the palm-tree from a sanitary standpoint, appears in this volume for the first time. It must come as a revelation to many that the palm-tree is so closely bound up with the life of man. One would almost think that if man and the palm-tree were left alone in the world he would still be able to house, clothe and feed himself in comfort, and live in a world which was still beautiful.

The foregoing is merely an outline of the contents of this most interesting and fascinating volume. No one who takes it up will find it dull—it is the very reverse of this. It is full of lessons for everybody interested in the welfare of mankind, and the motto of the book may be said to be that of the tireless Scarab, "Weary not in well doing," and illustrated in the charming little picture at the end of the entomological sermon.

The book is beautifully produced and fully illustrated. Many of the pictures are from the author's own photographs, taken during his sanitary wanderings in many theatres of war. The mere perusal of these alone is an education in itself. The Wellcome Bureau of Scientific Research is to be congratulated on being able to offer to the public, especially at this difficult time, such an interesting and instructive volume.

War against Tropical Disease: being Seven Sanitary Sermons addressed to all interested in Tropical Hygiene and Administration. By ANDREW BALFOUR, C.B., C.M.G., M.D., B.Sc, F.R.C.P. (Edin.), D.P.H. (Camb.), Director-in-Chief, Wellcome Bureau of Scientific Research. Published for the Wellcome Bureau of Scientific Research by Baillière, Tindall & Cox. Price 12s. 6d. net.

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CORRESPONDENCE.

"THE ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE."

The following letter is published for the information of Fellows :—

402819.

Home Office,
Whitehall, S.W. 1.
9th June, 1920

SIR,

I am directed by the Secretary of State to inform you that he has laid before the King your application of the 13th April last for permission to use the prefix "Royal" in the name of "The Society of Tropical Medicine and Hygiene," and that His Majesty has been graciously pleased to command that the Society shall be known as "The Royal Society of Tropical Medicine and Hygiene."

I am Sir, your Obedient Servant,

A. J. EAGLESTON.

The President, The Society of Tropical Medicine and Hygiene,
11, Chandos Street, Cavendish Square, W.

THE EDITOR, TRANSACTIONS OF THE SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

DEAR SIR,

It may prove of interest to those studying pellagra and the so-called deficiency diseases to briefly refer to results obtained in the treatment of a group of pellagrins, which were the subject of two papers by myself in the TRANSACTIONS of this Society.[1]

In 1912 were published some preliminary observations on pellagra occurring among the inmates of the Central Prison, Nyasaland, this being the first record of pellagra in Africa, apart from Egypt and the few cases observed by SANDWITH on Robben Island.

During 1913 the incidence of the disease was very much greater, and, in the second communication, the disease as then seen was dealt with at greater length from the clinical side, a number of observations bearing on the etiology of the affection were given, and the following statement made: "There are many reasons for supporting the possibility that pellagra is a deficiency disease . . . all 'maize' theories of causation as such are disproved." This conclusion having being arrived at, the next task was to effect an

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alteration in the prison diets, which reference to previous papers will shew were ill-balanced and deficient in accessory food factors. A new scale of diet, which included maize, rice, green vegetables, beans, an oily fish (dried), and meat, was eventually put into effect, and proper cooking arrangements were provided.

Unfortunately, with the outbreak of war, I had to leave Nyasaland, and the exigencies of the military position prevented the previous records being kept up. I have recently, however, had the opportunity of communicating with Dr. ELDRED, the medical officer in charge of the Central Prison, during the war, up to the end of 1919, and he has informed me that since 1914 no new cases of pellagra had occurred among the inmates of the Central Prison, and that many of the early cases improved or were completely restored to health.

Excepting the change in diet all the other conditions remained the same, so that this small group of men, under exactly known conditions, have constituted a practical feeding experiment, and the results go far to confirm the hypothesis that an ill-balanced diet is an etiological factor in the causation of pellagra.

I regret that circumstances do not permit me to support these findings with analyses of the several dietaries concerned

The actual part played by the deficiency of accessory food factors in "deficiency diseases" is, I believe, not nearly such a simple problem as many writers would lead one to think. I believe it is very necessary to keep clearly before one the attitude that "the absence of something cannot produce anything." Rather should we consider the problem presented by any deficiency disease as one in which, in the absence of some controlling factor (the accessory food factor), other factors come into play.

We should not be content, as there is a tendency to be, to label such and such a disease a deficiency disease, but we should seek further and attempt to find the toxic substance which is directly responsible for the lesions. Such a substance may be one normally produced in the body, or the result of abnormal metabolism, or one produced as the result of an infection by some living virus, but one which remains "undetoxicated" in consequence of the absence of an accessory food factor, acting, possibly, by an upset of the balance of endocrinic secretion.

KINNIER WILSON[2] has published a preliminary paper on the pathological findings in the nervous system of thirteen cases of pellagra (nine of which were cases from Nyasaland), which have a very direct bearing on the problem of pellagra etiology.

The changes described are comparable to those found in cases clinically diagnosed as subacute combined degeneration of the cord, and WILSON has shewn that in pellagra the pathological evidence favours the idea that "an ascending toxic lymph stream invades the cord *via* the lumbar and dorsal posterior roots, and that the effects of the toxæmia appear either simultaneously in the nerves and cord or previously in the former, and that the toxin is probably of alimentary origin."

Yours faithfully,

HUGH S. STANNUS.

1. "Pellagra in Nyasaland." H. S. STANNUS. *Trans. Soc. Trop. Med. and Hyg.* Jan., 1912. Vol. V. No. 3.

Ibid. Nov., 1913, Vol. VII. No. 1.

2. "The Pathology of Pellagra." S. A. KINNIER WILSON. *Trans. Neur. Sect. Roy. Soc. Med.* Jan., 1914.

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TRANSACTIONS

OF THE

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

JUNE 18TH, 1920.

VOLUME XIV. No. 2.

Proceedings of a Meeting of the Society held on Friday, June 18th, 1920, at 8.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Professor W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P. (*President*), in the Chair.

The PRESIDENT read the letter received from the Home Office published in the last issue, page 16.

ANNUAL GENERAL MEETING.

The adoption of the Report of the Council was moved by Dr. BAGSHAWE and seconded by Dr. PROUT and was carried.

Dr. A. BALFOUR presented the Treasurer's Report and Balance Sheet, the adoption of which was carried.

Dr. BEDDOES and Dr. MANSON-BAHR were reappointed Auditors.

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ORDINARY MEETING.

RECENT WORK ON ROUNDWORM INFECTION.

By F. H. STEWART, M.A., D.Sc., M.D.

I.—Geographical Distribution and Clinical Importance of Ascariasis in Man.

The parasite to which I invite your attention this evening is cosmopolitan in geographical distribution. It is also of very frequent occurrence, especially in the tropics and subtropics, in which a large proportion of the population have been found to be infected. In Europe it is pre-eminently a disorder of country districts. Leuckart, in 1876, estimated the percentage of children infected between the ages of three and ten, exclusive of those living in great cities, at 50 per cent. A more recent figure is from an examination of several thousand Italian soldiers in Venice, 22·4 per cent. It is extremely common throughout the Near East. In Cilicia, a prominent Armenian physician told me that during normal times almost 50 per cent. were infected, but as the result of the hardships of war the degree of infection had risen to nearly 70 per cent. O'Connor's figures for healthy Egyptians give 27 per cent. In India and Burma, percentages recorded vary from 5 to 27; in Indo-China, 18 to 60; Hong Kong, 47; China, 63; Corea, 85; and the Marianne archipelego, 93. Among natives in Africa, 4 to 20 and over. In North America, in the Southern States of the Union, ·3 to 42·8; in the West Indian island of Grenada, 70 to 85; these figures, which are only a few chosen from the bulky literature, will convey an idea of the frequency of the animal.

Turning to clinical effects, I will not take up your time with the literature, but merely give you impressions received from cases in my own personal experience, since I imagine that you would prefer personal observations, however incomplete, to summaries of published literature with which you are probably as familiar as I am. I have seen a few cases simulating severe malaria; others with anæmia so intense that the hæmoglobin was reduced to 30 per cent.; others in a typhoid condition; but the average case is one of moderate anæmia with lassitude, dyspepsia, and some muscular wasting. Taking into consideration the great frequency of the parasite, the amount of invalidity and the loss of working power in the world at large from this cause must be very great. It is also probable that the occurrence of the parasite in nations which habitually live at or near starvation level, may precipitate the occurrence of disease of which it is not the primary cause, such as beriberi, pellagra, and tuberculosis.

There is also a great economic loss from the occurrence of this parasite in domestic animals, especially in pigs. A very rough estimate gives a loss of £3,000,000 worth of food annually in the United Kingdom alone.

Some important work has recently been done on the mechanism of disease production by nematodes, especially by Ascaris. Bedson, in the *Annals of the Pasteur Institute*, found inflammatory changes in the suprarenal glands after the injection of minute doses of the perivisceral fluid of *A. megalocephala*. Shimamura and Fujii, in 1917, isolated from *A. lumbricoides* and other nematodes an albumose-peptone, which on injection into

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animals produced symptoms resembling those of Ascaris infection. More recently BENJAMIN SCHWARTZ, in the United States (1919), found that if *A. lumbricoides* are kept in salt solution, the solution acquires hæmolytic properties after twenty-four hours. The hæmolytic property is inverse to the presence of oxy-hæmoglobin which is found during the first twenty-four hours.

Apart from toxin secretion, it is probable that *A. lumbricoides* affects the host by mechanical injury to the mucous membrane of the intestine and by sucking blood. The oxy-hæmoglobin found by SCHWARTZ is, doubtless, derived from the blood of the host. Various conditions of injury, such as punctures, raised urticaria-like plaques and ulceration, have been described.

II.—PREVIOUS LITERATURE ON THE LIFE-HISTORY OF THE WORM.

Having thus very briefly and imperfectly considered the importance of the disease from the point of view of public health, I will approach my main thesis, namely the mode of infection and the life-history of the worm.

The literature can be divided into three groups: (1) the work of older zoologists—SCHUBART, RICHTER, DAVAINE and LEUCKART, (2) that of the pathologists and clinicians—MOSLER, LUTZ, GRASSI, CALANDRUCCIO and EPSTEIN, (3) recent work by myself, by RANSOM and FOSTER and by YOSHIDA.

The development of the egg in the free condition in the outer world was traced by SCHUBART, RICHTER and LEUCKART, beween 1850 and 1860. The segmentation and the formation of the embryo can occur in the outer world only. The upper layers of damp soil constitute the most favourable nidus. Moisture, a temperature above 60° F., abundant supply of oxygen, and absence of putrefaction are essential.

The further history has been more difficult to trace. There are clearly three alternatives: (1) hatching and some degree of development in the open, (2) hatching and development in an intermediate host with migration to the definitive host, (3) hatching and complete development in one host only.

DAVAINE excluded the first possibility. He proved that ripe eggs when swallowed by rats or dogs will hatch in the intestine, and he concluded, not unnaturally, that the further development occurred in one situation, namely, the small intestine of the appropriate host.

But, however probable this appeared, it did not turn out to be easy of proof. DAVAINE himself failed with an experiment on the ox. LEUCKART, the prince of experimental helminthologists, also failed to secure direct infection in a large series of experiments with ripe eggs of the appropriate species of Ascaris on man, pig, horse, cat and dog. He, as a zoologist, shewed that the problem was a wide one: that man did not stand alone in suffering from these parasites, and that the road of comparative parasitology gave the greatest promise of arriving at the solution of the problem. For, in truth, adult ascarids occur in every vertebrate form from fish upward, and encapsuled larval ascarids in vertebrates and invertebrates as well. These encapsuled larvæ are difficult to reconcile with a simple, direct life-history. They led LEUCKART to postulate an alternation of hosts. He suggested that larvæ encysted in fish developed into the adults in seals, porpoises, seabirds, and in carnivorous fish; that larvæ in the mole were destined to develop in the intestines of birds of prey; that those in the perivisceral space of insects would become adult in insectivorous mammals or birds. Such suppositions brought the facts of nematode life-history into parallelism with those of cestodes and trematodes. Indeed, there were only two alternative explanations of the facts: (1) alternation of hosts, and (2) the theory that these larvæ had wandered and lost themselves in inappropriate hosts—a theory previously held regarding all helminth larvæ, but which had been overthrown by KÜCHENMEISTER as regards cestodes.

The second group of workers—MOSLER, LUTZ, GRASSI, CALANDRUCCIO and EPSTEIN—experimented on man. Out of eleven experiments, four gave positive results, young or adult worms being passed after anthelminthics, at periods from one to three

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months after feeding. In estimating the value of these experiments, the high frequency of natural infection must be borne in mind.

In the experiments of MOSLER and LUTZ it was observed that dyspnœa, with bronchitis and fever, occurred some days after the administration of the eggs. This fact was not understood at the time, but, in view of recent additions to our knowledge, is of very great interest.

As the result of these experiments, the life-history of *A. lumbricoides* was considered as demonstrated. Medical and zoological text-books agreed that the worms were introduced into the definite host as embryos enclosed in eggs. Contaminated uncooked vegetables or dirty fingers were regarded as the most common vehicles. There was no suggestion of migration in the host. This was the condition of knowledge up to 1916.

III.—RECENT WORK ON THE LIFE-HISTORY OF ASCARIS LUMBRICOIDES.

During 1914 to 1916 I was stationed at Hong Kong, in medical charge of the Indian garrison. These troops were infected in a high degree with Ascaris, and the methods of infection, as described, did not appear sufficient to account for this. I therefore took up the investigation of the life-history.

My first experiments were directed to ascertain whether infection could take place through the skin, as in ankylostomiasis, since it appeared possible that, by contamination of the men's feet, eggs might be rubbed into the soft skin between the toes, and might there hatch either spontaneously or by mechanical violence. With this in view, I rubbed ripe cultures of eggs into the thinner portions of the skin of two young pigs, and after varying intervals cut out small portions of skin. Serial sections shewed many free embryos on the skin surface, but none had penetrated.

I then fed the two young pigs on large numbers of ripe eggs, in both cases with negative results. Thereafter I repeated DAVAINE'S experiments on the feeding of rats. Live and free larvæ appeared in the fæces from twelve to twenty-four hours after the feeding, thus confirming DAVAINE'S results; but six days after the first feeding a further development took place in the experiment: all the four rats which I employed became ill, their respirations were rapid, there was some blood-stained discharge from the nostrils, and one rat died. On dissection, the lungs were found intensely congested, and when portions were teased out in salt solution, many nematode larvæ in active movement made their appearance. Larvæ of similar character were found in the liver.

I will not weary you with accounts of further experiments which I performed on rats and mice. It will be sufficient to give the results, which are that ripe eggs introduced into the alimentary canal of these animals hatch within the first twenty-four hours, probably in the small intestine. The larvæ issuing from them bore into the wall of the intestines and migrate to the liver in forty-eight hours, probably by the portal veins. They are found in the liver in the capillaries of the interlobular zone, and appear as if arrested at those points where the branches of the portal vein are dividing into capillaries. About the same time they appear in small numbers in the lungs, and by the fifth day in large numbers, and in sections are found to be lying in the alveoli surrounded by masses of red-blood corpuscles. It is, therefore, probable that they reach the lungs in the blood-stream, and act as emboli in the pulmonary capillaries. On one occasion I have found a larva in the heart-blood on the fifth day. The number of larvæ in the lungs increases up to the seventh day, and, if the infection is massive, the lungs are solidified by the hæmorrhage into the alveoli. A dose of five to ten thousand ripe eggs will produce symptoms of pneumonia in a mouse or rat.

Larvæ pass up the bronchi on the sixth and seventh days, and appear in the trachea on the seventh and eighth. I have found them in the trachea up to the thirteenth day. On the ninth day they begin to descend the alimentary canal, and, passing rapidly through the œsophagus, stomach and small intestine, accumulate in the cæcum, colon and rectum. They begin to pass out in the fæces on the tenth day, and have been found in the cæcum and fæces up to the fifteenth day.

During the migration through the liver, lungs and intestine the larvæ undergo great

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development; they increase in size from ·28 mm. at hatching to 2mm. in the trachea and intestines. The muscular system and the lateral membranes (swimming organs) make their appearance during the passage through the bronchi and trachea, and while traversing the alimentary canal the cuticle becomes thickened and hardened. The larva as found in the cæcum swims actively in S-shaped curves.

This completes the life-history in the rat and mouse. My observations up to this point were published in the *British Medical Journal* during 1916, and in *Parasitology* in February, 1917, and January, 1918. At that time, having failed to produce infection by the eggs in pigs, and also in guinea-pigs and dogs, I looked upon the rat and mouse as the only animals in which larval development could take place, and I consequently announced these rodents as the intermediate hosts of the worm. I considered that infection of the definitive host, man or pig, took place by the ingestion of larvæ liberated in the fæces of the rodents. That this announcement was at least premature my own work very soon shewed. For, having secured assistance from the Government of Hong Kong, I was able, in the beginning of 1917, to experiment on sucking pigs instead of on weaned animals over two months old; and I then found that in these young animals the larval development of the worm could take place in exactly the same manner as in the rat and mouse. A dose of 50,000 eggs would certainly produce a fatal pneumonia in a sucking pig, and 20,000 would cause a very severe attack.

These results obviously put an entirely different complexion on the problem. Since larval development could take place in the host of the adult worm, it only remained to trace the transition from the larva in the lung to the adult in the intestine. In any experiment it might be assumed that larvæ were present in the lung if signs of pneumonia occurred about the seventh day; to a less certain degree it might be assumed if the culture used had previously being found infective to mice.

I performed four such experiments in Hong Kong, in March, 1917, and some adult worms were found in two of the four pigs killed on the twentieth and thirtieth days, but there was a reasonable doubt as to whether the worms were the result of the experiment. The experiments were performed in great haste while I was on the point of transfer from Hong Kong to India. The results were published in the *Indian Medical Gazette*, August, 1917, and in *Parasitology*, January, 1918.

I did not obtain an opportunity of returning to the subject until I found myself stationed in Cairo, in the summer of 1918. Here, through the kindness of Dr. KEATINGE, Director of the Cairo Medical School, I was introduced to a Greek gentleman, who on an estate near Mataria, bred pigs in considerable numbers. This gentleman most generously allowed me to experiment on some young animals. In these new experiments I decided that in view of the frequency of worm infection in pigs, the only satisfactory procedure would be to feed a number of young pigs and to kill them at short intervals, beginning with the stage at which I had arrived in rodents, namely the fourteenth day. Owing to lack of time, however, I was only able to proceed with two animals. Both had severe pneumonia on the eighth day. One was killed on the fourteenth day, and numerous larvæ measuring up to 4 mm. were found in the intestine. The second pig was killed on the nineteenth day, and no worms were found. This negative result in an animal which eleven days previously undoubtedly had thousands of larvæ in its lungs is remarkable.

At this point my experiments were interrupted.

Summarising the results so far, the larval development described with the migration by the vascular system through the liver to the lungs, and again into the intestine occurs in rodents, in pigs, and in man also, as is clear from the experiments of MOSLER and LUTZ on man in which pulmonary symptoms were noted. The transition from the larvæ in the lungs to the adult in the intestine has not been traced completely as yet.

Subsequent to the publication of my work on rats, mice and pigs, the subject was taken up by RANSOM and FOSTER, of the United States Department of Agriculture. They repeated my experiments on rats, mice and pigs with similar results, and they also found that the same development would take place in guinea-pigs and rabbits. They also infected a lamb and a young goat. In the former they found worms of nearly adult size three months afterwards, and in the latter, worms of 10 mm. after twenty-seven days.

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They claim that these successes in animals which do not frequently harbour Ascaris established the hypothesis of direct infection in one host only. They are certainly very valuable evidence. On the other hand, they have repeatedly failed to secure the appearance of adult worms in pigs. Therefore, although it is becoming more and more probable that infection is direct, it is still necessary, in my opinion, to follow the transition from larva to adult closely. Until this has been done no one can assert that further surprises are not in store, and, in dealing with an animal of so frequent occurrence in nature, outside infection can never be completely excluded.

YOSHIDA has still more recently published two papers on this subject, in March and September, 1919.

He redescribes the migration of the larvæ in guinea-pigs and rabbits, and finds that it will also take place in monkeys. He considers that the larvæ do not travel through the vascular system, but through the serous cavities, and directly through solid organs. He has proved that the larvæ possess remarkable powers of migration. He applied an infected liver as a poultice to the nape of the neck of a guinea-pig, and shewed that larvæ later on appeared in the lungs of this second animal.

One highly important practical point remains to be decided, even assuming that full development can take place in one host, namely, Can infection also take place from larvæ passed in the fæces of rodents? YOSHIDA and I have both shewn that larvæ taken from the body of one animal can infect another, but this, of course, is a condition of affairs which would not readily come about in nature, except in the case of carnivorous animals such as dogs and cats. But, if the larvæ in the fæces retain their vitality, they could readily gain access to new hosts. That some nematode larvæ can resist desiccation is shewn by the cases of *Anguillula tritici*.

DISCUSSION.

The PRESIDENT: Colonel STEWART is to be congratulated on the success which has attended his very interesting and important experiments. He has shewn the way in which these ascarids migrate, and that is an advance on what was known before. Possibly, if he had had opportunities, he would have carried the matter much further. It is a great pity that he had to move away from Hong Kong to India, and so his experiments were interrupted. It is to be hoped that he will have further opportunities of carrying on this important research. It is very gratifying to find British workers leading in helminthology. We have the work of Professor LEIPER and Dr. ATKINSON at Shanghai, then Professor LEIPER's successful researches into bilharzia in Egypt, and now we have this work of Dr. STEWART. Their success is a source of gratification to this Royal Society, of which they are fellows.

Dr. R. T. LEIPER: I join you, sir, in words of congratulation to Colonel STEWART. It must have been the intention of the Council of the Society, in inviting him to give the first paper after this became a Royal Society, to indicate their high appreciation of his work. Colonel STEWART is continuing his researches. We shall follow with great interest the further outcome of his experiments.

The problem which Colonel STEWART raised with regard to the intermediate host is one which, apparently, has not yet been finally settled. I am inclined to agree with him that the life-history may not be a simple one exactly like that of the ankylostoma. May it not prove that for *Ascaris lumbricoides* man and the pig are intermediate hosts as well as definitive hosts, in a sense somewhat similar to the tapeworm, *Hymenolepis nana*? It seems to me that the first migration of the embryo through the blood-stream, after being set free from the egg, may be taken, perhaps, to correspond with the feeding-stage of the ankylostoma before it undergoes that metamorphosis which makes it truly infective. In the ankylostome, this stage takes place outside the body; in the Ascaris, within the host.

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Can Colonel STEWART tell us if he has tried to infect the same hosts through the skin or through the mouth with the larval ascarids after these have passed through lungs and gut? Such an experiment would prove if Colonel STEWART was originally right in considering his experimental animals to be intermediate hosts. They may be definitive hosts as well.

Colonel CLAYTON LANE: I would like to add a word of appreciation of Colonel STEWART's work which he did as an I.M.S. man—work outside his own duties. That has usually been the fate of I.M.S. men, that the work they have done for the advancement of medicine has been, so to speak, an "extra."

As regards this particular subject, there seems to be no doubt that there is some very essential factor of which we are ignorant. The work which RANSOM and FOSTER have done in it (to which the author alluded, but which has only just been received in this country) shews an almost entire failure to infect pigs with the ripe eggs of the Ascaris, so that direct infection is entirely unproved, and we seem back at the beginning. RANSOM and FOSTER have also shewn the extraordinary degree to which the eggs resist drying, so it seems possible that they are air-carried. If so, there arises the question of entry into the body after hatching. RANSOM and FOSTER consider that nobody knows the conditions which facilitate hatching. I suggest that if eggs are air-carried, they may be inspired and hatch on the nasal mucous membrane, the larvæ entering the circulation in that way. They may, that is, be hatched out and reach the lungs without first passing through the liver. I think that the passage through the liver, with its extra set of capillaries to negotiate, must be a very big handicap, and that, therefore, the question arises as to whether that is the normal route. Ascaris eggs are certainly very susceptible to light currents of water (in levitation, ascaris eggs are carried away when hookworm eggs stay on the slide). It seems to me that those bosses upon them which produce the resistance to water-currents will equally produce resistance to air currents, and facilitate carriage by air. At all events, their great resistance to drying merits further consideration in this connection. With regard to YOSHIDA's conviction that larvæ travel directly, instead of through the circulation, RANSOM and FOSTER have found larvæ in the blood of the portal vein and in the blood of the pulmonary artery, so it looks very much—and I think other things shew it too—as if they are carried to the lungs in the circulation.

Mr. H. A. BAYLIS: Although I am a stranger to this Society, I would like to draw attention to a certain point about other ascarids, which throws a little sidelight on the question of intermediate hosts. Dr. STEWART referred to certain larval forms of ascarids which are found encysted in the peritoneum and connective tissues of fishes, and I think it is possible, on morphological grounds, to assume that the adult forms of these larvæ are certain forms which are found in seals and in fish-eating birds. I think that supports the view that *Ascaris lumbricoides*, which belongs to the same family, may very well have an intermediary host, although the forms in question belong to a rather specialised group. Colonel STEWART referred to the excretory cell in the larva, and I am acquainted with his work on one of those other larval forms where he described this excretory cell. In the adult forms the same type of excretory cell persists, and they are characterised by having certain cæcal appendages of the intestine or of the œsophagus, or both, and many of them have a peculiar modification of the hinder end of the œsophagus. One does not find these structures in *Ascaris lumbricoides* or *Ascaris megalocephala*. The classification of the ascaridæ is not on a very satisfactory basis, and the majority of species are still referred to the genus Ascaris. But, although their relationships need much further investigation, I think the presence of an intermediate host in the cases referred to is a point in Colonel STEWART's favour.

Major E. E. AUSTEN: I would be glad if Colonel F. H. STEWART, when he replies, will say whether by chance he paid any attention to the possibility of the *house-fly* playing a part in the dissemination of ascariasis. As he is aware, J. O. SHIRCORE, at Mombasa, found Ascaris eggs, in a few instances, in the intestine of the common house-fly; and it

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seems to me that the author's remark that ascariasis in Europe is pre-eminently a disorder of country districts possibly has some bearing on the point, because if the house-fly is in any way concerned in disseminating these parasites, it is obviously in the country districts of Europe that it would find its opportunity.

Lieut.-Colonel STEWART (in reply): I thank you, Sir, and the other gentlemen who have spoken on the paper, for the very kind references to my work. I was under exceptionally favourable circumstances for it, and so was able to achieve results where other people have failed. The climate of Hong Kong favoured the development of the eggs, the material was very abundant, and one could always get large quantities of worms, both from man and the pig. I was able to get much assistance from the medical authorities of Hong Kong, especially the late Dr. MACFARLANE, who was in charge of the bacteriological laboratory of the colony.

In reply to Dr. LEIPER, I have performed experiments with larvæ from fæces. I imagine that if larvæ from the fæces are infective at all, they would be equally so from the pig, or from rodents. All three might act as intermediaries. I have recently performed some experiments with larvæ from the fæces of mice administered to pigs, but I have not been able to get more advanced forms in the pigs I fed with it. The experiments were difficult to carry out, because the number of larvæ one can get in the fæces of the mouse is very small. If one gives large doses, the animal dies, whereas if one gives only a small number one can recover only a few larvæ. There is a risk of mutilating the larvæ by trying to extract them from the fæces before administering to the pig. The young pig spits out much of what one gives, so that the number of worms recoverable from a given area of intestine would necessarily be quite small.

Colonel CLAYTON LANE suggested the possibility of infection through dried eggs being carried in the air. My experience in the drying of eggs is contrary to that of certain others. I have found that when eggs were dried the embryos died. There may be different races of worms adapted to different climates. Hong Kong has a humidity going up to 90 per cent., and the worms found there may not be adapted to dry surroundings. I do not think the route through the tissues is a handicap to the animal; it gives to it an opportunity to undergo development necessary for its establishment in the intestine. The larva develops its cuticle and its excretory cell, and passes from an extraordinarily unprotected animal to one which can withstand the rather trying conditions it must meet with in the intestine, subject to the play of the digestive juices.

With regard to the ascarides of fishes, of course the excretory system of *Ascaris decipiens* is intermediate between that of the larval encysted nematodes and the excretory system of the more advanced forms which inhabit mammals.

Major AUSTEN referred to the possibility of the house-fly carrying these worms. There is no doubt it can carry them mechanically, but I do not think the house-fly is in any way essential in the life-history. I do not think anyone has ever found any development in the eggs after they had passed through flies. When I began experimenting in Hong Kong, I thought the fly must play some part. Owing to the primitive nature of sanitation in the colony, accumulations of fæces swarming with maggots are common. I performed many experiments with maggots in Ascaris-infected fæces, to see if they would take up the eggs, and whether developing forms would be found in them. But I found no eggs, nor traces of development. I think the explanation of country districts being more infected is that there is more opportunity there for fæcal infection. In large cities the sanitary arrangements are better, and the fæces are not distributed through the upper layers of the soil, where eggs can undergo the development necessary to make them infective.

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REVIEW.

MALARIA AT HOME AND ABROAD.

One of the chief features of this useful and attractive book is the clear and simple way in which it is written. It has made its appearance at a very opportune moment, and will undoubtedly be welcomed by those for whom it is intended—medical men and administrators who may have to deal with malaria at home or abroad during peace or war. The author's long and varied experience has well fitted him for his task, and it is at least doubtful if anyone else at the present time could thus happily have summed up our knowledge of malaria, as it exists both in England and Wales and in foreign, and more especially tropical, countries.

The work commences with a definition of malaria and a general description of its specific pathogenic agent, enhanced by a well executed graphic scheme shewing the position of the human malaria parasite in a zoological classification. For those not familiar with protozoological nomenclature, it would have been helpful if the derivation of words like trophozoite and merozoite had been given, but the account of the life-history of the parasite is carefully written, and here, as in the second chapter—which deals with factors concerned in the spread of malaria—a useful summary is forthcoming. In this second chapter there is a note on the occasional absence of male gametocytes in the blood of malaria carriers—a point often forgotten—and mention is made of the failure of GRASSI and others to convey the disease by anophelines fed upon such patients. A suggestive diagram illustrates the etiological chain, composed of the three chainlets representing respectively source of infection, carrier of infection, and recipient of infection, each linked together, and each containing its own links. The author shews how this chain may be broken, and discusses in an interesting manner, and with special reference to the work of CHRISTOPHERS, how the various factors influence the development and spread of malaria. The rôle of large warm-blooded animals—a subject specially studied by ROUBAUD in France—is briefly considered, an instance being cited where non-immune soldiers lived with malaria-carrying soldiers in a room over a stable in which cows were kept. They did not become infected, although anophelines were numerous in the stable, partly because the mosquitoes found in the blood of the cows a more accessible food supply than that of the soldiers.

The section on the technique of blood examination is good, though the thick-drop method is relegated to the chapter dealing with diagnosis; and it is better when preparing an ordinary blood film by the needle method, to ensure that both margins of the film are some little distance from the edges of the slide instead of only one of them, as shewn in Fig. 12. This can be secured by using a small drop of blood.

It was possibly a mistake to exclude the account of fluid or "fresh" blood specimens from this section, for, while it is true that in routine diagnostic work stained preparations are almost invariably used, yet, in the tropics, conditions not infrequently necessitate the examination of the fresh blood, and it is a great advantage to be thoroughly familiar with the appearance of the malarial parasite in such blood.

The sections dealing respectively with the examination of the film, the identification of the species of parasite, and its discovery and study in the insect host, form an admirable exposition of the subjects with which they deal. There follows a very well illustrated and complete chapter on mosquitoes. It contains a great deal of information, is well up to date, and is written in a manner which cannot fail to commend it to the medical entomologist, be he a tyro or the reverse. Mention is made of *Harpagomyia genurostris*, a mosquito that has the curious habit of living in close association with ants, which supply it from their mouths with honey they have collected. This is instructive, in view of the relationship existing between ants and certain aphides.

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Little is said about *Anopheles plumbeus* as a vector of malaria. Presumably the work done at the Liverpool School of Tropical Medicine in connection with this species is so recent that it could not be included. It will, no doubt, figure in a second edition.

One of the most interesting and important chapters in the book is that which details the essential features of a malarial survey, and discusses the epidemiology of malaria in England. The work carried out under the auspices of the now defunct Local Government Board and of the Ministry of Health is described, and there is a very instructive map of England and Wales, shewing the distribution of ague in the nineteenth century, the localities where locally contracted cases of malaria occurred in 1917 and 1918, and the very numerous places from which imported relapsing cases were reported between March 1st and November 1st, 1919. A study of this map impresses one with the importance which malaria has assumed in this country, and the need for such a work as that under review.

Very fittingly, the next chapter considers the same subjects in the tropics, and also discusses malaria from the endemic standpoint in hot countries. Spleen rate and parasite rate are defined, and if the observations are rather limited to India and Ceylon, this is, doubtless, because these are the regions with which Colonel JAMES is specially familiar, and where most of his valuable work has been accomplished.

The seventh chapter is a long one, for it is concerned with the symptomatology, pathology and diagnosis of malaria, but it is conveniently divided into four sections, devoted respectively to a clinical account of ordinary cases, further clinical details and pathology, deaths from malaria, together with accounts of the disease in children, blackwater fever, etc., and diagnosis. It will be seen that a somewhat original method has been followed, and thereby the reader is the gainer, especially as regards the third section, which enters fully into some questions such as certain complications and sequelæ, not always adequately discussed in the text-books. To some of them, indeed, as, for example, psychical affections, the experiences of the late war have directed special attention.

Stress is laid on the marked differences between the symptomatology of endemic and epidemic malaria, while the series of temperature charts illustrating section one of this chapter has been well-chosen, inasmuch as they are taken from ***untreated*** cases, and hence the disturbing influence of quinine upon the temperature curve is eliminated.

The following interesting conclusions on cases in soldiers returned from Macedonia are here quoted, as there has been much faulty reasoning as regards these cases, and the author's views appear to be well founded. He says—

> *As regards the great majority of the cases*, the following conclusions seemed to me justifiable :—(1) Their primary infection had undoubtedly been with heavy doses of sporozoites ; to this I attribute chiefly the large numbers of parasites usually to be found in the peripheral blood, and the frequency of the relapses. (2) There was no evidence that either the parasites or the human host had become habituated to quinine, but much evidence that the quinine treatment which the patients had received had been very efficacious in controlling the manifestations of the disease. This was shewn by the comparatively small degree of anæmia, the absence of splenic enlargement, and other obvious differences between the condition of these cases and that of the untreated cases which have been described above. (3) By the time they arrived in England, which in many cases was about eight months after their primary infection, most of the patients already shewed markedly that the infection was being overcome. This was clear, both from the instances of spontaneous cure of relapses and from the observation that ten grains of quinine given each morning sufficed, in the great majority of cases, to reduce the frequency and severity of relapses in an unequivocal manner.

Colonel JAMES is perhaps inclined to belittle overmuch the ill-effects of chronic malaria and to base his belief on experience in India, where at one time confusion existed between chronic malarial cachexia and kala-azar. It is true that the disease tends towards recovery, but in cases of repeated infection and reinfection the condition of the patient is not infrequently graver than he indicates, especially when the liver has been profoundly affected. Such cases are distinct from the acute primary cachexia described in section three.

One cannot here consider the whole of this chapter in detail, but it is a succinct account of the symptoms, pathology and sequelæ of malaria, and includes in a small compass all the really important recent work on these subjects.

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Blackwater fever occupies a sub-section by itself; its symptomatology, diagnosis, prevention and treatment being considered together. The author does not discuss the vexed question of etiology. All will not agree with him that quinine should be withheld during the attack.

The section on diagnosis is very helpful and full of wise advice. The statement is made that the thick-drop method of blood examination should be employed only to supplement the ordinary thin film method, and never as a substitute for the latter. It ought, however, to be noted that the thick-drop method was exclusively employed with much success for the diagnosis of East African malaria in South Africa. By its means also the chance of finding parasites in blackwater cases is increased by something like 50 per cent. and though it is not a good method for distinguishing the type of parasite present, save as regards crescents, many would be inclined to term it the method of choice, provided they were certain of their reagents. It is, however, true that it must be thoroughly mastered before being adopted in routine work, and it finds its chief application where a great number of cases have to be examined in a short time.

What may be called the "specific" treatment of malaria is alone dealt with, and to its consideration Chapter VIII. is devoted. If one may say so, it is characterised by a great deal of common-sense, and should be hailed with joy by the perplexed practitioner, who must often have wondered why so many different ways of giving quinine existed. The treatment of the acute attack is clearly differentiated from the after-treatment. The author states that the former should terminate after five days, but not a few believe that, to get the best results, the initial treatment of 30 grains a day should be prolonged for a fortnight or three weeks. This, however, is a matter of opinion, and there is much to be said for lessening the dose as quickly as possible, provided the result desired is not thereby endangered.

Amongst the causes preventing quinine from acting efficiently, poor preparations of the drug and lack of absorption might have been mentioned, and it would have been interesting to have had the author's opinion of the statement that quinine does not act well if albumin is present in the urine, a condition necessitating a strict milk diet.

Nowhere has the vexed question of quinine prophylaxis been better handled, and those who oppose this method cannot do better than read Chapter IX., which also deals with the treatment of "carriers." Section two of this chapter gives "Practical rules for carrying out methods of prevention and eradication." A little more might have been said about repellents for mosquitoes, and, under "hand-catching," the value of soaping the hands might have been mentioned as a practical point. These, however, are small matters, and, generally speaking, the rules are excellent. The duties of medical practitioners as regards the discovery and treatment of cases in England and Wales are duly noted.

The last part of the volume is devoted to "Mosquito Control," and it is not the least interesting and instructive. The advice given is essentially sound and practical. One would like to have seen sanitas-okol mentioned in addition to cyllin, for it is a reliable larvicide, and has been largely used; while something might have been said about cresol, as it is so easily obtained and so effective. Again, there might have been a reference to the part played by river steamers in the tropics as breeding places and disseminators of mosquitoes, and the best way of dealing with them, but, as the author states, his book does not pretend to be a complete treatise. The wonder is that a volume of its size and type is so complete. With pardonable pride, Colonel JAMES states that it has received the warm approbation of Sir PATRICK MANSON. That in itself is sufficient praise, and it is praise well merited, for, apart from the qualities already mentioned, the work is no mere collection of views on malaria, but largely a record of personal experience embodying the author's well-considered and well-matured views with respect to a subject on which, as has been said, he is specially qualified to give an opinion.

ANDREW BALFOUR.

Malaria at Home and Abroad. By S. P. JAMES, M.D., D.P.H. (Lieut.-Colonel I.M.S., retired). London; John Bale, Sons, & Danielsson, Ltd.

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CORRESPONDENCE.

THE EDITOR, TRANSACTIONS OF THE ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

SIR,

I have just read with deep interest Dr. G. D. H. CARPENTER's paper, published in the *Reports of the Sleeping Sickness Commission* of the Royal Society, No. XVII., 1919, and two important points suggest themselves to me.

Dr. CARPENTER suggests the "provision of attractive artificial 'pupa shelters' as a means of reducing the flies' numbers," and he has shewn that this can be effected to some purpose, no less than 295 pupæ being collected in one day in a "pupa shelter."

He, furthermore, found by experiment that in "pupa shelters" in which half the floor surface was bare and the other half strewn, either with leaves or flakes of bark or small logs laid on the surface, a much larger proportion of pupæ was deposited on the strewn surface than on the bare.

On p. 100, par. 2, he states: "Moreover by catching the adult fly only a small percentage of females (rarely greater than 25 per cent., often less than 10 per cent.) is destroyed."

May it, therefore, not be of interest to utilise these "pupa shelters" as traps, by smearing with some adhesive substance small logs, propped on supports, a couple or three inches off the ground, so that the female Glossina when alighting on a log, prior to larvipositing, may become adherent and so destroyed?

I believe that it may also prove of interest to know whether if some non-volatile poisonous chemical, such as arsenite of soda, the presence of which the female fly would be unconscious of, was incorporated in suitable quantity with sand, and spread over the surface of a "pupa shelter" to a depth of an inch, pupæ deposited in such a shelter would hatch out.

If the latter or both these methods are productive of results, a distinct reduction of the numbers of Glossina should be achieved. The "pupa shelters" would not only act as they do at present, but would also be automatic and almost permanent destructors of fly, and the urgent necessity for regular frequent visits for the collection of pupæ could be extended to longer periods.

I am, Sir, yours faithfully,

J. O. SHIRCORE.

Dar es Salaam,
27th May, 1920.

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TRANSACTIONS

OF THE

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

OCTOBER 15TH, 1920.

VOLUME XIV. No. 3.

Proceedings of a Meeting of the Society held on Friday, October 15th, 1920, at 8.45 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Professor W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P. (*President*), in the Chair.

RESEARCH INTO THE TRYPANOSOMIASIS PROBLEM: A CRITICAL CONSIDERATION OF SUGGESTED MEASURES.

BY PROFESSOR WARRINGTON YORKE, M.D.

Six years have elapsed since the subject of trypanosomiasis was last discussed by this Society. During this period comparatively little has been done on the subject, but since the war ended several very important papers have appeared, and further investigations are now contemplated by both the French and ourselves. I feel, therefore, that it is now opportune to review the present position, and consider briefly the direction which further research should take.

If we refer for a moment to the position shortly before the war, we remember that as a result of a considerable polemic which arose out of certain observations made by KINGHORN and myself in Northern Rhodesia, and by the Royal Society Commission in Nyasaland, the Secretary of State for the Colonies appointed an Inter-Departmental Committee, the terms of reference of which were as follows :—

To report—

(1) Upon the present knowledge available on the question of the parts played by wild animals and tsetse-flies in Africa in the maintenance and spread of trypanosome infections of man and stock.

(2) Whether it is necessary and feasible to carry out an experiment of game destruction in a localised area in order to gain further knowledge on these questions, and, if so, to decide the locality, probable cost, and other details of such an experiment, and to provide a scheme for its conduct.

(3) Whether it is advisable to attempt the extermination of wild animals, either generally or locally, with a view of checking the trypanosome diseases of man and stock.

(4) Whether any other measures should be taken in order to obtain means of controlling these diseases.

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This Committee examined numerous witnesses, and considered written statements from various foreign experts and others who were unable to attend in person. In May, 1914, it issued a report, the general conclusions of which are as follows :—

Knowledge of the disease, its cause, and its remedies, is still in the making, and hasty and imperfectly considered action of a drastic character, such as the attempt to effect a general destruction of wild animals, is not justified by the evidence before your Committee. On the other hand, your Committee recommend that until direct means of checking the fly have been discovered, the food supply of the fly and the chances of infection should be lessened in the vicinity of centres of population and trade routes by the removal of wild animals, and that for this purpose freedom be granted both to settlers and natives to hunt and destory the animals within prescribed areas and subject to prescribed conditions.

So far as regards the disease in Uganda, the measures already taken have effectually checked the epidemic, and removed the mass of the population from the danger of further infection. While, no doubt, it is desirable that the land lying near Victoria Nyanza should be rendered again available for the use of that population, this is not a question of immediate urgency, and may well await the acquisition of further knowledge.

With regard to the Nyasaland and Rhodesian form of the disease, its incidence on the population is slight, and it is not increasing. The evidence points to its being an old disease, endemic and not extensive, and though it is unsafe to prophesy, there is no apparent reason to anticipate its appearance in an epidemic form. Having regard to the importance of the question whether man forms a reservoir of the human trypanosome, your Committee would lay emphasis on the desirability of further experiments, as suggested in paragraphs 41 and 42 above. (See also Appendix D.)

It must be recognised that the evidence all points to the conclusion that, if tsetse-fly could be eliminated or removed from contact with human settlement, sleeping sickness would practically disappear, infection conveyed by other biting flies being a negligible factor in the spread of the disease.

For this reason your Committee attach great importance to a proper and sufficient equipment of entomological research into the bionomics of the incriminated tsetse-flies. This form of research has, in their view, been insufficiently pursued up to the present time. The workers have been zealous, but few in numbers, and the work consequently limited to only a very small portion of the fly-belts and areas from which the danger arises.

Different views are taken as to the prospect of dealing with the fly, but it was, as your Committee think truly, said by more than one of the witnesses that in this form of research there is a large element of chance—that accident may at any time lay bare a secret which may lead to the solution of the problem—and that the multiplication of workers is the multiplication of those chances.

Your Committee think, therefore, that, within reason, there should be devoted to this form of inquiry a considerable portion of such funds as may be available in British Possessions, and that endeavours should be made to obtain the co-operation in this work of Foreign Powers in their African Possessions, the results of the work being from time to time tabulated and collected.

Research will, no doubt, be continued as to the nature of the different trypanosomes, and the part they play in the infection of man or of domestic stock.

The proposed experiment of removal of wild animals from a selected area may produce valuable results, both as regards knowledge of the habits of the fly, and as to the extent to which the infectivity of the fly, and subsequently the infection of man or stock, is derived from the wild animals.

As has been pointed out, the result of this experiment cannot be confidently anticipated. There are possible fallacies and uncertainties involved from the very nature of the problem, and in dealing with natural conditions there is always the possibility of unknown factors vitiating or defeating action based on the apparent results of any such experiment.

Nevertheless, your Committee think that there is sufficient to justify an expectation of useful results, and they recommend that if a suitable locality can be found, where an experiment can be carried out at a reasonable cost, it should be undertaken. They are, however, of the opinion that the carrying out of the other measures recommended should not be delayed pending the results of the experiment, which cannot be expected to emerge for two or three years.

It is perhaps needless for your Committee to say that they hope that medical research as to treatment of the disease and the production of immunity will be continued.

The above recommendations relate mainly to the acquisition of knowledge on which further action may be based. As regards immediate action, your Committee strongly recommend that measures of clearing should be undertaken where they are practicable and would tend to check the spread of the disease, and render life in settlements and travel by road safe for men and stock.

This question has been fully dealt with already, and it is only necessary to say here that your Committee attach much importance to this matter.

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Your Committee would point out that the action in various directions which they have suggested is for the general benefit of every part of Africa in which tsetse-fly exists, and is or may be the cause of sleeping sickness, so that so far as the cost would fall on local funds they should not fall only on that Possession which may be selected for any particular experiment, as being that in which the knowledge sought is most likely to be attained. It is even possible that Foreign Governments might be willing to share in the cost, but this is hardly a matter on which it is fitting that your Committee should make any substantive recommendation. Nor is the question of a contribution from public funds one for them to deal with, though it is, they think, doubtful whether local funds would be sufficient to bear the costs of the various inquiries and experiments which they think desirable.

Action on this report was necessarily deferred owing to the outbreak of war; other work and other diseases monopolised attention, and research into trypanosomiasis practically ceased. It has now become apparent that the ravages of sleeping sickness have increased during the war in certain of the French equatorial colonies, and also in portions of the Belgian Congo. This knowledge caused the Société de Pathologie Exotique to appoint a committee, under the presidency of M. LAVERAN, to consider what means should be taken to deal with the situation. This Committee reported in July to the Société, and its report was accepted and forwarded to the Colonial Minister, and also to the Governors-General of the French Colonies in Africa. About the same time our Secretary of State for the Colonies took action on the report of the Earl of Desart's Inter-Departmental Committee, and asked the Imperial Bureau of Entomology for recommendations. The matter was referred by the Bureau to its Glossina Sub-Committee, which subsequently issued a report containing certain recommendations for future action.

Now, the different procedures adopted in France and in this country seem to me to be a matter not without interest to this Society. In France the Société de Pathologie Exotique took action and advised their Government; in this country the Government has to seek for advice, and does so, not from this Society but from the Imperial Bureau of Entomology. One cannot help feeling that herein lies a moral which this Society should take to heart. Surely on matters relating to tropical medicine, in all its various aspects, a Society like ours, which contains amongst its hundreds of members, medical men, veterinarians, entomologists and zoologists from all quarters of the earth, should be able to crystallise its ideas, and advise the Government accordingly. If this Royal Society is to be a living force, and not a mere agglomeration of academic pedants, it should be able to function in this manner on questions of great medical and economic importance.

In this paper I propose to refer briefly to the report of the Société de Pathologie Exotique, and then to discuss in more detail the report and recommendations of the Glossina Sub-Committee of the Imperial Bureau of Entomology. It is important to realise at once that the problems which confront us are, in many respects, different from those with which the French have to deal.

In the French equatorial colonies the most urgent need of the moment is the control of the epidemics of *gambiense* sleeping sickness, which are ravaging certain parts of them. The primary prophylactic measure recommended by the French is the atoxylisation of the sick, with the object of destroying the flagellates in the blood of all infected cases. Marvellous results are to be anticipated from this procedure, provided a sufficient personnel be available for some years in all infested districts, so that all the infected could be recognised and treated. With reference to measures directed against the tsetse, the report states that one can hardly dream of causing the complete disappearance of the Glossina, which swarm on the banks of most of the streams of equatorial Africa, but measures can be taken to remove the natives and to protect them against bites. Clearing has been declared by all observers one of the most useful measures which can be practised. There is room for research to ascertain whether we cannot destroy Glossina by opposing them with other insects or animals which are their natural enemies. It is recalled that pigs are particularly attractive to Glossina, and it is suggested that possibly herds of these animals, and of other resistant beasts, could be grouped at certain places in the vicinity of the villages in such a manner as to form a sort of protecting screen. As the spread of sleeping sickness is due to the incessant movement of porters and travellers, and as the disease is aggravated by want, excessive porterage, and fatigue, strict administrative control is necessary. The manner in which the above recommendations should be applied, and the organisation necessary, are discussed in detail at the end of the report.

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Although in none of the British colonies is there at the present time any very serious epidemic of *gambiense* sleeping sickness, we have in Central and South-Eastern Africa to deal with a situation, which in its way is just as serious, and at least as difficult of solution, as that which confronts the French in their equatorial colonies. Fortunately, the amount of *rhodesiense* sleeping sickness occurring in Central and South-Eastern Africa is at the present time small, but the question of trypanosomiasis of domestic stock is one of the greatest economic importance. This fact is clearly indicated in the opening paragraphs of the report of the Glossina Sub-Committee, which reads as follows : –

> Some idea of the importance of the tsetse-flies of the *Glossina morsitans* group as an obstacle to the development of Tropical Africa may be gathered from a consideration of the wide areas over which they range. In Southern Rhodesia, in which the fly-belts are comparatively small and have been more carefully surveyed than elsewhere, their extent has recently been estimated at 9,000 square miles. With reference to North-Eastern Rhodesia, Messrs. KINGHORN and MONTGOMERY have expressed the opinion that it would be difficult to find a continuous area of fifty square miles free from *G. morsitans* anywhere except on the Serenje plateau, around Fort Jameson, and on the high plateau between Lakes Nyasa and Tanganyika. In Nyasaland probably more than a fifth of the area is infested by tsetse-flies ; while in Tanganyika Territory the fly-belts must cover nearly half the country.

The Report of the Glossina Sub-Committee consists of three portions : the first summarises very briefly the present position as regards the various tsetse-flies ; the second gives a list of suggestions for observations and experiments ; and the third makes detailed recommendations concerning the organisation necessary for the elucidation of the foregoing problems. It is to the second and third portions of this report that I desire to draw your attention.

It is impossible here to give a list of all the suggestions for observations and experiments, but they deal with the following points : (1) the distribution of the fly, (2) the effects of clearing, (3) breeding-grounds, (4) food of the fly, (5) destruction of adults, (6) parasites, (7) enemies of the fly, and (8) influence of odours. It will be observed that the subject which was the main one before the Earl of Desart's Inter-Departmental Committee in 1913-14, and which was really the cause of the appointment of that Committee, viz., the inter-relationship of game, tsetse, and the pathogenic trypanosomes of man and domestic stock is completely ignored. This is the more remarkable as the report of the Glossina Sub-Committee was called for directly as the result of the report of the 1913-14 Inter-Departmental Committee. However, I will not go further into this matter at the moment, but proceed to discuss the methods by which the Glossina Sub-Committee propose to investigate the problems which they themselves have decided it is desirable should be solved. The following are the recommendations :—

> For the elucidation of the foregoing problems it seems desirable that there should be at least six different experimental stations, widely distributed over Tropical Africa, and the countries that suggest themselves as being most suitable for the purpose are Zululand, Southern Rhodesia, Nyasaland, Tanganyika Territory, the Sudan and Northern Nigeria.
>
> The sélection of the precise areas in these countries that would afford the best facilities for these investigations would require a more intimate knowledge of local conditions than the Sub-Committee at present possesses. But it may be laid down generally that the various areas should be representative of different types of environment, and that they should, as far as possible, be situated in localities in which the presence of tsetse-flies is actually obstructing settlement, or is likely to do so in the near future.
>
> Each of these stations should be under the control of a competent entomologist, and he should have at least one assistant who would be able to carry on the work in the event of the senior man falling sick or going on leave. Unfortunately, at the present time there are very few men who combine the requisite qualifications and experience for this class of work. This difficulty could be met by starting, in the first instance, say, two stations only. In these the new men could be concentrated for training purposes. After a time, the most promising pupil could be left in charge, and the others would proceed to open up a new station elsewhere.
>
> While the observers in each station would be expected to keep an alert watch on all aspects of this complex problem, it would probably be advisable for some of the stations to give special attention to certain lines of work, such as control by clearing, artificial breeding-places, rearing of parasites, etc.
>
> Each station ought to send in a monthly progress report to the Imperial Bureau of Entomology, so that the Glossina Sub-Committee may be kept advised of all the latest developments. They

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would thus be in a position to co-ordinate the work, to prevent overlapping, and to arrange promptly for the testing under divers conditions of any new hypothesis that may be put forward.

As the work progresses, it is probable that special lines of inquiry will become necessary, for which the aid of a protozoologist or veterinarian may be required, and provision should be made for such eventualities.

It will be observed that what, in brief, the report recommends is (1) that there shall be set up at least six different experimental stations in widely-separated places in Tropical Africa; (2) that each of these stations should be under the control of a competent entomologist, who should have at least one assistant who would be able to carry on the work in the event of the senior man falling sick or going on leave; and (3) that each station should send in a monthly report to the Imperial Bureau, which would then be in a position to co-ordinate the work

Now the sum of £50,000 (if such a sum can be raised) has been mentioned as the cost of this organisation over a period of five years. A brief calculation will, however, shew that such a sum, which allows for an annual expenditure by each experimental station of £1,700, is totally inadequate for the purpose. Such an amount is hopelessly insufficient to provide for salaries, transport, camps, labour, equipment and leave of the personnel of the Commission; there will be no balance for the purpose of experiment and investigation, to say nothing of the many other incidental expenses inseparable from such undertakings, and the possibility of any experimental work requiring the employment of labour is completely excluded.

The first criticism I have to make, therefore, is that if the above recommendations are adopted, a much greater sum than £50,000 must be provided.

Let us, however, assume for the moment that money is a matter of no consequence, and proceed to consider whether such an organisation as this is likely to succeed in obtaining the information which will enable the Imperial Bureau to answer the questions which it enumerates within a period of five years.

It is admitted that "unfortunately at the present time there are a very few men who combine the requisite qualifications and experience for this class of work." In order to get over this difficulty it is proposed to start with only two stations and concentrate the "new men" in these for training purposes. Now the task of training these "new men" would obviously be a lengthy one; ill trained men who were ignorant of the literature of the subject, which is already rather extensive, and who know neither Africa nor the native or his language, could hardly be expected to advance knowledge of this most complex subject very much. If four of the proposed stations, therefore, require to be staffed by "new men," nothing very valuable in the way of information is to be anticipated from them for a number of years.

Again, if we assume that this difficulty can be surmounted and sufficient well-trained entomologists can be found within, say, a year, to provide each of the six experimental stations with its full staff of one competent entomologist and at least one assistant, would such an organisation prove adequate to the task which is required of it? If allowances be made for such contingencies as sickness and furlough, it is clear that during a considerable proportion of the five years there would be in each station only one entomologist—a condition of affairs not far removed from that which has obtained during the past ten years.

Realising these facts, we must ask ourselves whether it is probable that the solution of the problems enumerated in the report would be obtained within any reasonable space of time by such an organisation. Let us for a moment consider several of the more important problems which it is proposed that the one or two men in each of these widely scattered stations are expected to investigate:—

1. What are the precise factors that determine the limits of a fly area?
2. In the open "orchard bush" so much frequented by *G. morsitans* is it sufficient to clear away shrubs and branches of trees to a height of twelve or fifteen feet, or must trees of twenty to thirty feet be felled?
3. What is the minimum width of cleared belt that would effectively check the spread of the fly?

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4. Can the number of fly be materially reduced by judicious burning of grass at times when food is scarce and shelter limited?
5. Compare the results obtained by indiscriminate clearing and by the clearing of breeding places after these have been defined.
6. Test the possibility of introducing additional parasites of different kinds from remote parts of Africa.

These are doubtless important questions, and their solution and that of the others enumerated in the report might enable us to formulate some scheme for dealing with the problem, but what I wish us to consider is whether it is reasonable to expect that one or two entomologists, unable by lack of sufficient funds to dispose of more than a very limited amount of native labour, are likely to succeed in providing answers to them.

My general criticism of this portion of the report is, then, that the organisation which is recommended is entirely inadequate for the work which it is expected to perform. The solutions of the very questions which the report states require elucidation can be obtained only by intense and concentrated effort and not by the adoption of such a scheme as the one we have just considered, which involves dissipation of energy and precludes possibility of experimental investigation on a large scale. The adoption of the measures recommended in this report will result in failure not only to elucidate any practical solution of the problem, but to advance knowledge in any considerable degree; the report is, in fact, a proposal to perpetuate as regards research the state of affairs which existed before the war.

These remarks, however, do not complete my criticism of the report of the Glossina Sub-Committee; as I have already stated, the main question which was before the Earl of Desart's Committee, viz., the relation of game, fly, and trypanosomiasis of man and domestic stock is completely ignored. The work which immediately preceded, and which was the direct cause of, the appointment of Lord Desart's Committee demonstrated that the main, and for practical purposes the only, reservoir of the pathogenic trypanosomes of man and domestic stock is the big game. So far as I am aware, nobody has criticised the results obtained by KINGHORN and myself in the Luangwa Valley, and by the Royal Society Commission in Nyasaland, in so far as they relate to the pathogenic trypanosomes of domestic stock. Now it is important to recognise that, judged from the economic point of view, trypanosomiasis of domestic stock is at present in Central and South-Eastern Africa immeasurably more important than is trypanosomiasis of man. On this account alone it seems to me that the effect of game elimination in a definite zone should be decided once and for all by a carefully designed and controlled experiment.

The observations by KINGHORN and myself that 16 per cent. of the game in the Luangwa Valley were infected with *T. rhodesiense*, the trypanosome pathogenic to man, although quickly confirmed by the Royal Society Commission in Nyasaland, met with the most hostile criticism on all hands. While most of this criticism was based on hypotheses and speculation, and was therefore of no value, some of it was founded on scientific observations, and requires careful consideration; I shall refer to it later.

The question whether game really forms the main reservoir of the trypanosome pathogenic to man is not of mere academic importance, as a glance at the report of the Conference on Sleeping Sickness, held at Pretoria on 9th March, 1920, will shew. This Conference was called by the Minister of Public Health in connection with an application to the Union Government by the Natal Coast Labour Recruiting Corporation, Ltd., for the removal of the prohibition in respect of recruitment of native labourers for the sugar estates from the country between the Limpopo and Zambesi Rivers—also with the recently reported extension southward of tsetse-fly and the infection of sleeping sickness. Unfortunately, time does not allow one to record in detail the findings of this Conference. The following are, however, among the most important.

It was unanimously agreed that it would be dangerous to allow recruiting for the Natal Sugar Estates in the belt of country between the Limpopo and the Zambesi Rivers, or in any area where there is any risk of an infected native being recruited.

In regard to the question, "What are our present southern limits of sleeping

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sickness infection, and what are the risks of the introduction of the disease into the Union?" it was decided by the Conference that under the present conditions the risks of introduction of infection were undoubtedly very great, and that both as regards the Zululand-Mozambique border and infected areas in Rhodesia, the existing arrangements for safeguarding against introduction of sleeping sickness are totally inadequate.

After discussion, it was agreed :—

> That the Zululand-Mozambique border should be closed for all native movement inwards, except at two ports of entry, namely, Nduma and Maputa—this not to interfere with the existing system of visiting passes for local natives. Medical examination at the border would be very difficult to enforce, and would be unreliable even if supplemented by a microscopic examination of the blood. A prolonged period of medical observation, with blood examinations and animal inoculations, is necessary in order to exclude the disease.

This resolution is followed by others relating to the strengthening of police patrols on the border the provision of penalties for infringement of regulations, and the issuing of certain additional regulations.

I have ventured to refer to this report at such length because it bears most directly upon the subject under discussion. It will be seen that the resolutions are of a rather drastic character, and interfere in a greater or less degree with the economic life of the community.

Now the deliberations of this Conference and the resolutions it passed are based on the assumption that man constitutes the main reservoir of the trypanosome which affects man in these localities, viz., *T. rhodesiense*. If this be not so, and the game be the main reservoir of the virus pathogenic to man, then the above recommendations, with the resulting economic disturbance and the expenditure involved in putting them into force, are futile.

I will now review briefly the evidence on which those who deny the identity of the human parasite with that of the same appearance found in game base their contention.

(1) *Epidemiological.* It has been asserted that these two parasites are undoubtedly different; because in certain *morsitans* areas, the animal trypanosome (*T. brucei* vel *T. ugandae* vel *T. pecaudi*) is found in game and stock and in tsetse-fly without cases of human trypanosomiasis occurring.

(2) *Experimental inoculation of the human subject with the game trypanosome.* As it is upon these observations that most faith is placed, it is necessary to refer to them in detail. In 1913 TAUTE fed upon himself, with negative result, laboratory-bred *G. morsitans* which had been rendered infective with the *rhodesiense*-like game trypanosome; he also subsequently inoculated himself with 2 cc. of the blood of a dog naturally infected with the same trypanosome, and here again the result was negative; these experiments were carried out at Lubimbinu, in Portuguese East Africa. Much more impressive, however, is a later series of experiments performed in 1919 by TAUTE and HUBER. They inoculated themselves and 129 natives, many of whom were in poor condition from malaria and other causes, with the *rhodesiense*-like trypanosome from four naturally infected horses and two mules. In no instance was an infection obtained in man, although the animals (rats, dogs and a goat) used as controls all became infected and died. Naturally, the greatest importance has been attached to these experiments, which by many are regarded as conclusive in shewing the human and game parasites are not the same.

While I am quite prepared to admit that at first sight this conclusion may seem correct, I think, if we consider the facts more closely, it will become clear that KLEINE and TAUTE have not proved their point, and that their observations are quite in harmony with the view that the two trypansomes are identical. To my mind the various facts which have been elicited regarding human trypanosomiasis in South Central Africa can best be explained on the view that the game is the great reservoir from which man (as well as his domestic animals) becomes infected. To develop my argument, I find it necessary to commence with the postulate that man is extremely resistent to infection with any member of the genus Trypanosoma under natural conditions, or, in other words, that he possesses great natural immunity to infection. This postulate is, however, no

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mere baseless hypothesis, but is supported by considerable evidence. In view of the fact that man has never been found infected with *T. congolense*, and only on a single occasion with *T. vivax*—pathogenic trypanosomes which are exceedingly common and of universal distribution in Tropical Africa—it is evident that man must possess a considerable degree of natural immunity to them. Again, certain facts regarding *T. gambiense* are in harmony with this postulate. If man be readily susceptible to infection with *T. gambiense*, it is difficult to account for the absence of sleeping sickness in an epidemic form in the West Coast Colonies and in certain other parts of Tropical Africa. The cause of the epidemic in Uganda has recently been discussed by DUKE, in a most able paper, and explained by him on the ground of a mechanical transmission by *G. palpalis* from man to man of a strain of *T. gambiense* of increased virulence. It is a well recognised fact that passage of *T. gambiense*, or of any other African trypanosome, in the laboratory, from animal to animal greatly increases its virulence for that species of animal; for example, the strain of *T. gambiense* maintained by us in Runcorn was at the time of its isolation from the human being of very slight virulence for rats, these animals living, on an average, over a hundred days, but after the strain had been passed through these animals for a number of years its virulence for them had so increased that they died in about fourteen days. This increase of virulence is, of course, artificial and does not take place when the parasite is passed alternately through the vertebrate and invertebrate hosts; but, as DUKE has pointed out, an analogous increase in virulence probably also takes place in nature when conditions are favourable—that is, when there is a broad and intimate contact between man and fly. Such conditions were found in the densely-packed canoes on the fly-infested shores of Victoria Nyanza, the fly acting the part of the syringe in the laboratory and conveying infected blood mechanically from one native to another.

To return now to *rhodesiense* sleeping sickness, how can we explain its distribution otherwise than on the assumption that man possesses great natural immunity? The more its distribution is investigated, the more does it become apparent that the infection is distributed more or less uniformly over an enormous stretch of country in Central and South-Eastern Africa. Now, if infected man were the reservoir from which other men become infected, one would naturally expect to find a more or less considerable focus of the disease in every locality where an infected case is found. This, I think, is not so; the total number of cases of *rhodesiense* infection which have as yet been discovered is still rather small, but the figures, such as exist, suggest that the disease is widely and uniformly distributed, allowances, of course, being made for differences in the density of *G. morsitans*. I hold, therefore, in direct contradistinction to KLEINE, that what epidemiological facts we have at our disposal suggest that man becomes infected from the widely diffused game reservoir rather than from other infected men.

Turning now to TAUTE's remarkable experiments on the human subject, let us consider what exactly he has proved. In my judgment, all that TAUTE has proved is that man is very resistant to infection. I believe that if, in his first experiment, TAUTE had fed his laboratory-bred flies on an infected man, instead of on monkeys and antelopes, he would have obtained a similar result; whereas if, in the second experiment, his inoculations had been made from the blood of an infected man, instead of from infected horses and mules, the result might well have been different, because of the exaltation in virulence of the strain for the human being, which might manifest itself as the result of passage through two human hosts consecutively without an intermediate passage through the invertebrate host. TAUTE's results, far from militating against the hypothesis that the game is the reservoir from which man is infected, are only what would be expected if the hypothesis is correct. KINGHORN and I shewed that in the Luangwa Valley one in five hundred flies was infected (in an infective condition) with the *rhodesiense*-like trypanosome, and similar figures were afterwards obtained by the Royal Society Commission in Nyasaland. Under these circumstances large numbers of human beings must be bitten every day by an infective fly, and yet cases of human infection are very few. The only conclusion we can draw from this is that man is remarkably resistant to the trypanosome in question; consequently, on the assumption that the game trypanosome is the same as that found in man, many more than 130 persons would have to be used in experiments

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such as that conducted by TAUTE and HUBER before one could expect to obtain a positive result.

Before leaving the subject, I should like to draw attention to another aspect which has apparently been entirely overlooked by those who, like KLEINE and TAUTE, deny the identity of *T. rhodesiense* with the trypanosome of the same appearance found by KINGHORN and myself in game. It is clear that a large proportion of the game must be infected with *T. rhodesiense*. The Royal Society Commission in Uganda, in 1910, succeeded in infecting all of eleven antelopes by feeding upon them laboratory-bred *G. palpalis*, experimentally infected with *T. gambiense* from a case of sleeping sickness. FRASER and DUKE (1912) found that the antelopes so infected remained in perfect health for over a year, and DUKE later recorded that he was able to infect *G. palpalis* from an antelope infected experimentally with *T. gambiense* twenty-two months previously. WECK (1914) records that in German East Africa he inoculated successfully an antelope with blood from a human being infected with *T. rhodesiense*; the antelope shewed no signs of disease, but its blood was infective for monkeys and dogs five weeks later, and there appears no reason for doubting that the blood of this antelope would also have infected *G. morsitans*; nor is there any ground for doubting that if antelope can be infected directly from the human being, they could also be infected from one another. This being the case, it follows that antelope must become infected with *T. rhodesiense* one from the other by *G. morsitans* precisely in the same way as they are infected with the common pathogenic trypanosomes of domestic stock.

I maintain, therefore, that the game constitute the main reservoir from which *G. morsitans* draws the trypanosomes pathogenic to man and his domestic stock. For practical purposes the game constitute the only reservoir, because man, domestic stock, monkeys and the small vermin rapidly die of the infection, whereas the antelope are tolerant, and harbour the parasites in their blood presumably for long periods without exhibiting signs of disease.

There seems to me, therefore, to be a clear indication that any investigation which is contemplated with a view to enable us to deal with the problem of trypanosomiasis in South and Central East Africa should have as its primary objective the obtaining of precise information concerning the inter-relationship of game, fly, and the trypanosomes pathogenic to man and stock.

Information on two questions, both of great importance, might be expected to emerge from a carefully conducted and controlled experimental elimination of game from a prescribed district. These are :—

1. The effect on the tsetse-fly.
2. The effect on the trypanosomiasis of man and domestic stock.

May I at this point refer for a moment to the great game drive which took place in Zululand in August, 1920, wherein 500 hunters are said to have taken part, and to have been aided by an army of 5,000 natives acting as beaters. The area comprised in this drive is stated to be the size of Cornwall and Devon. Unfortunately, I have no information regarding the organisation of this great drive, beyond the brief reference which has appeared in the daily press, but I should like to point out that, unless it was preceded by a thorough and scientific investigation of the conditions, both in respect of fly and of the trypanosomes of game, man and domestic stock which existed before the drive, and unless it is followed by an equally careful investigation extended over a sufficient length of time, we shall have no precise information regarding the results of what must have been an unparalleled slaughter of game. I sincerely trust, therefore, that those who organised this drive had this fact in mind, otherwise the failure to take advantage of an unrivalled opportunity would be deplorable.

To sum up, I am of opinion that the report of the Imperial Bureau of Entomology is open to serious criticism, and that the recommendations contained in it should not be put into operation. I hold this view, firstly, because the report evades what I regard to be one of the main problems requiring immediate investigation, namely, the dependence of fly and trypanosomiasis on the game; and, secondly, because its recommendations concerning the method by which the problems, which the report itself considers to be of

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primary importance, should be investigated are wholly inadequate for the purpose, are little if anything in advance on the methods adopted in the past, and are calculated to result in failure at the cost of much money and time; in short, these recommendations appear to me to indicate a lack of appreciation of the gravity and difficulty of the problem demanding investigation.

In contradistinction to the recommendation of the Imperial Bureau of Entomology, I would suggest (1) that in future investigation effort should be concentrated instead of dissipated; (2) that the work of entomological and medical and veterinary research into the trypanosomiasis problem be combined under one central organisation in Africa, and that such organisation be supported by the pooled contributions of all African States interested; (3) that the personnel of the investigating commission be large enough to ensure continuity of work in all directions, thus obviating interruptions due to such exigencies as illness or leave, and preventing the staleness and inertia which are so likely to result from isolation; (4) that sufficient funds be placed at the disposal of the investigating commission to allow of the employment of adequate native labour, so that experimental work can be undertaken on a sufficiently large scale, thus enabling the investigation of the dependence of fly and trypanosomiasis on game, and of the various problems enumerated in the report of the Imperial Bureau of Entomology to be carried out in a satisfactory manner, and with some reasonable prospect of success.

Discussion.

The President: Professor Warrington Yorke has raised some very important issues in this valuable paper, and as there are a number of Fellows here who have had a great experience in regard to trypanosomes and sleeping sickness, I shall expect the contribution to give rise to considerable discussion.

Professor Yorke has shewn the serious situation of sleeping sickness from the epidemiological as well as from the economic standpoint, and he has strongly emphasised the important fact that, in any investigation which may be undertaken to discover the best methods of combating or preventing sleeping sickness, the inter-relationship of game, the tsetse-fly and pathogenic trypanosomes in man and in domestic stock must not be ignored. He complains—very rightly, I think—that this aspect of the question has not been considered sufficiently in the recommendations of the Bureau of Entomology. And he further points out that the organisation which is proposed is totally inadequate for the problems to be solved. I agree with Dr. Yorke's point of view; I do regard it as a most important thing that the inter-relationship I have mentioned should be thoroughly investigated; and I agree, too, that the organisation proposed could not possibly advance the question further than its present stage. I remember that when, in 1913, I went to Uganda, I was amazed to find that there were only two scientists working at the subject there; or perhaps I should say one scientist, for one was a lady, Miss Robertson, who had been working on the subject for some considerable time, while the other was an American, Mr. Fiske, who had just arrived, and this in a region where 200,000 deaths had taken place within a few years, and when, even at the date of my visit, there was a certain amount of anxiety with regard to this disease. On my return home I availed myself of the opportunity of submitting, in my general report, an expression of my astonishment at the inadequate means being taken to deal with the subject. I do not blame the Colonial Office, because they do not hold the pursestrings; they are held by the Treasury, and the Treasury are not interested in tropical diseases or in the health of the inhabitants of the tropics. Colonies which are rich have to take take care of themselves—and rightly so, as they are quite able financially to look after their own health Colonies that are poor have to do the same, and they suffer accordingly. It is in this uneconomical attitude that the difficulty lies in securing proper research and efficient health conditions.

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I remember an instance which illustrates the attitude taken by the Treasury in regard to tropical diseases. A meeting was held in the Mansion House for the purpose of obtaining funds for the London School of Tropical Medicine, and Lord HARCOURT, who was then Secretary for the Colonies, announced that, after some difficulty, he had been able to secure £500 a year for the Tropical School, but he added that this was not under any circumstances to be regarded as constituting a precedent. Sir GEORGE REID happened to be present, and he was the next speaker, and, in a humorous and somewhat sarcastic speech, expressed his pleasure at hearing that the grant was not under any circumstances a precedent, for he hoped that next year the amount would be £5,000, That is how it strikes other people. They see clearly we do not receive sufficient money for this scientific work, and that we do not treat the matter in a really serious way.

Professor YORKE has flattered this Society in making his suggestion that we might have been consulted in this matter, and I hope the Secretary of State may be induced in future to adopt the principle that, on the few occasions when there is something of great importance on which he desires to seek outside advice on epidemic diseases of the tropics, it would not be a bad thing to consult this Royal Society, for, as Professor YORKE has stated, this Society consists of medical men, of veterinarians, of entomologists, of zoologists, most of whom are familiar with the tropics, and, therefore, likely to be well qualified to give valuable advice on a broad and practical basis. There has been in the past, and still is, a tendency to work in compartments, but however useful it might have been in the past this system is not adapted to solve the problems of to-day. Under the present conditions it is desirable that the advice should be much more general, and that, instead of isolation, we should have co-operation.

I hope Fellows will give us an interesting evening, and perhaps some will criticise Professor YORKE's criticisms.

Dr. GUY MARSHALL: I have listened with the greatest of interest to Dr. YORKE's very able and instructive paper. But, while naturally one must pay the greatest attention to the opinions of a man of his experience in these questions, it would be an act of insincerity on my part if I were to pretend that he has won from me either agreement with his views or approbation of his methods.

As a member of the Glossina Sub-Committee of the Imperial Bureau of Entomology, on which he has poured his scorn, I hope to be able to shew that the picture of its incompetence which he had prepared for your delectation is overdone. In the first place, he has drawn a contrast between the action of our own Government and that of the French Government in regard to the trypanosomiasis problem. Apparently the head and front of the offending of the Colonial Office is, that they consulted the Imperial Bureau of Entomology on a purely entomological question relating to tsetse-flies, although that Bureau was especially created by the Colonial Office to advise on entomological matters. Nor, I think, can that department be fairly charged with neglecting the medical and protozoal aspects of trypanosomiasis, for in the past they have freely sought the advice of the Royal Society, whose Tropical Diseases Committee comprises a number of the leading authorities on Tropical Medicine. I do not know whether the author really thinks that this Society ought to set up a rival organisation to that Royal Society Committee, but that is a matter upon which I do not care to express an opinion.

Professor YORKE has unwittingly given an entirely erroneous complexion to the whole aspect of the Glossina Sub-Committee's report. After setting out some of the proposals which were made by this body, he draws attention to the fact that we have entirely ignored the main subject which Lord Desart's Committee was appointed to consider, and he says, "This is the more remarkable as the report of the Glossina Sub-Committee was called for directly as the result of the report of the 1913-14 Inter-departmental Committee." I can assure him that that statement is absolutely incorrect; there is no shadow of truth in it. The matter that led to the appointment of the Glossina Sub-Committee was purely an entomological one, and it arose in this wise. The Principal Medical Officer of one of our African Colonies was asked by his Government to make arrangements for entomological investigations in connection with Glossina, and he was told that a certain sum

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would be available for them. He replied that he had no entomologists on his staff, that he saw no opportunity of securing the services of one, and that, moreover, the sum named was quite inadequate for accomplishing any really useful work. He, therefore, suggested that it would be better if amounts, voted by various colonies, could be pooled, and used for entomological work under a central control. This letter was sent to me from the Colonial Office for a report. I supported the proposals with some modifications, and my memorandum was referred to the Committee of the Imperial Bureau of Entomology, who appointed a Sub-Committee to deal with it. The matter had no connection whatever with Lord Desart's Committee. Nevertheless, it is hardly fair to say that we have ignored the recommendations of that body, for they specially emphasised the need for more work along entomological lines in the following words: "Your Committee attach great importance to a proper and sufficient equipment of entomological research into the bionomics of the incriminated tsetse-flies. This form of research has, in their view, been insufficiently pursued up to the present time." There is no doubt that it has been unduly neglected in the past, and the suggestions of our Glossina Sub-Committee were framed in the hopes of being able to remedy that defect. The criticisms levelled against their report are really quite premature, for their proposals are merely tentative and preliminary, and not a cut-and-dried scheme. We have to remember that the whole matter is dominated by the question of funds.

Professor YORKE has urged that the sum suggested as necessary for carrying out the work outlined by the Glossina Sub-Committee, namely, £50,000, is inadequate for the full accomplishment of the programme. That is, no doubt, true; but in these matters reasonable discretion is necessary, and it would be foolish to start by making demands that to the layman might appear excessive. It seemed, therefore, wiser to ask for a smaller sum with which to make a start, and then, if we can shew good results, we shall be justified in seeking further assistance.

The more extreme advocates of wholesale game destruction appear to think that those who disagree with that policy do so because they refuse to admit that there is any vital connection between tsetse-flies and game. But this is not so. If all the mammalia could be permanently eliminated from a given area, no one can doubt that the flies would disappear. But this would also be the case if all the shade upon which these flies are dependent were eliminated without killing the game. Now, these two lines of attack both suffer from the same drawback, namely, that they are only feasible in small areas, and cannot in practice be applied to wide stretches of country, such as N.E. Rhodesia, four-fifths of which is under fly. Dr. TAUTE, in his communication to Lord Desart's Committee, in 1913, pointed out the folly of trying to eradicate Glossina by such means, on a big scale, unless you can at once place settlers on the land, so as to maintain economically the conditions averse to the fly.

In regard to the use of shade destruction as a means of eliminating tsetse-flies, Mr. W. F. FISKE's excellent work on *G. palpalis* in Uganda has shewn that the maintenance of the fly population in a fly area is due to the existence of a number of foci presenting optimum conditions of food, shade, and breeding-places. It is not, therefore, necessary, as had been previously supposed, to destroy all the shade in a fly area in order to get rid of the flies (which is economically impracticable), but only the shade in the vicinity of these foci; and this discovery has rendered clearing operations much more feasible as an effective sanitary measure.

It seems not unreasonable to suppose that a more exhaustive knowledge of the life-history and essential requirements of *G. morsitans* may lead to a similar modification in current views as to game destruction as a means of controlling this insect. It may well be that we may be able to limit destruction to certain kinds of mammals only, and this only in certain limited areas or at certain definite seasons. For our present knowledge as to the essential factors that restrict the range of this species is so inadequate that most suggestions for its control are purely speculative. For a similar reason, it seems desirable that further entomological research should precede any argument in game destruction, otherwise the results of the experiment may be quite erroneously interpreted.

Professor YORKE has criticised the proposal of the Glossina Sub-Committee to

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establish experiment stations in a number of different parts of Africa; he desires that all efforts should be concentrated in one place. This arises from the fact that he is not attempting to consider the subject from an entomological standpoint, but has in view merely the inauguration of a game-destruction experiment in relation to *G. morsitans*. Our Sub-Committee, however, has taken a broader view of the tsetse problem. We have to consider that there are at least four distinct species of major importance, whose life-histories and idiosyncracies require much further elucidation, for we know that what is true of one species is not necessarily true of another. Moreover, in different parts of Africa the same species may occur under very different conditions, so that control measures that may be suitable in one place may be quite inapplicable in another. For example, the measures that were effective in absolutely eliminating *G. palpalis* from a small, entirely isolated and relatively populous area like the island of Principe, would be impossible to carry out on the shores of Victoria Nyanza, and still less so in Southern Nigeria.

From the entomological aspect, therefore, it is obvious that there is much research yet to be done in many different localities, and the more men that we can have at work simultaneously, the more quickly are we likely to secure the information that is necessary for the solution of this complex and important problem.

Major E. E. AUSTEN: There are one or two points to which I would like to direct the attention of Professor YORKE and the meeting, though, after Dr. MARSHALL's speech, there is little for me to say. As another member of the Glossina Sub-Committee I accept whatever share of responsibility falls to me on that account. But I would point out that, so far as I know, the Sub-Committee's report has never been published, in the sense that the Report of the Departmental Committee on Sleeping Sickness was published; unless I am mistaken, it is a private Memorandum, drawn up to formulate our own ideas, and to explain in general terms to those from whom we hope to be able to raise funds what we intend to do. Therefore, I support Dr. MARSHALL in deprecating criticism at this stage, since matters have not advanced far enough to warrant it.

As to the subject-matter of Professor YORKE's very able paper, it is quite true, as he has admitted, that in Central and South-Eastern Africa trypanosomiasis in domestic stock is an immeasurably more important problem than the disease in man, and that big game is the reservoir of the trypanosomes that are fatal to stock is no new discovery; we have known it ever since Sir DAVID BRUCE (then Surg.-Major BRUCE) carried out his epoch-making investigations in Zululand in 1895-6. The real point at issue, however, is involved in the fact that, while admitting that game is the reservoir of the trypanosomes which affect stock, and render many parts of Africa at the present time unsuitable for colonisation, it is yet denied by some people—who, I think, are competent to be regarded as authorities—that Professor YORKE is justified in declaring it to be established that game in those regions is the main reservoir not only of the trypanosome which is fatal to domestic animals, but of that which is so lethal to man, in other words, that these two trypanosomes are one and the same. There is—if I may say so without offence, and no offence is intended—a *trail of sleeping sickness* over this paper, and this feature has always been noticeable in the campaign against the game. Obviously, game as the reservoir of sleeping sickness is a proposition of vastly greater and more sinister import than game as the reservoir of trypanosomiasis of domestic stock, yet, before we sanction anything in the nature of wholesale game destruction, we need to be very sure of our ground. Professor YORKE tells us that the game is the main reservoir from which *G. morsitans* draws the trypanosomes of Rhodesian sleeping sickness. But Professor YORKE, while in the Luangwa Valley, Northern Rhodesia, acquired, at any rate, some evidence shewing that man does not always rapidly succumb to this parasite. In the "Final Report of the Luangwa Sleeping Sickness Commission of the British South Africa Company, 1911-12" (*Annals of Tropical Medicine and Parasitology*, Vol. VII., No. 2, June 10th. 1913), the authors (Professor YORKE, Dr. KINGHORN and Mr. LLEWELLYN LLOYD), dealing with *Trypanosoma rhodesiense* infection in man, write as follows (*loc. cit.*, pp. 186, 187):—"No exact data exist as to the duration of the disease in natives, but it would appear to be short. Many of the cases complained of no subjective symptoms of the disease when diagnosed, and

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presented very few objective signs, but in general they lived only a few months. Occasionally, however, a patient is more resistant, and one native definitely proved to be infected with *T. rhodesiense* is still alive and in a state of apparent good health a year later." Professor YORKE has told us to-night that, in his opinion, the result of the heroic experiment by TAUTE and HUBER "might well have been different" if the inoculations "had been made from the blood of an infected man, instead of from infected horses and mules," owing to the enhanced virulence of the parasite that might have been expected under such conditions. On the other hand, in the Luangwa Valley, as on the shores of Victoria Nyanza, infected blood may often be conveyed by tsetse-flies "mechanically from one native to another," a process which, as DUKE has pointed out, is not incompatible with increase of virulence on the part of the parasite. Professor YORKE himself will admit that he cannot have it both ways. If the trypanosome taken from man is more dangerous to man than that from animals, I venture to maintain that this native, who had trypanosomes in his blood, but was still apparently well at the end of a year, and may be alive now for all we know, is, if he be not under lock and key, far more dangerous to the surrounding inhabitants than very many antelopes. Dr. AYLMER MAY (Principal Medical Officer of Northern Rhodesia), in the course of his examination before Lord Desart's Committee, stated, in reply to questions put to him by the Chairman, that 40,000 natives in Northern Rhodesia had been examined for sleeping sickness, that the disease was not increasing there, and that there were fewer cases than in the previous year (120 in all in four and a half years, since 1909). I asked Dr. MAY in how many instances out of the 40,000 natives examined a blood examination had been made, and he replied, "Roughly, in about 20 per cent." In other words, while but 40,000 out of 100,000 natives in the sleeping sickness area were examined for sleeping sickness at all, it was only in the case of 8,000 individuals that an actual blood examination was made. Does Dr. YORKE believe that this examination of the blood of 8,000 out of 100,000 people is sufficient to prove that not one of the remaining 92,000 had trypanosomes in the blood? And does he maintain that his native, who was alive and well a year after being proved infected, was the only exception to the rule in Tropical Africa, and that he was so unfortunate as to come upon that single exception?

Professor YORKE, when himself examined before the Departmental Committee, and questioned about the result of TAUTE's original experiment, to which reference has been made to-night, would not admit that it proved anything more than that TAUTE himself was immune to the particular trypanosome concerned. But Professor YORKE proceeded to add: "If he had inoculated one hundred human beings the result would have been very different." Here, in this paper, we are told that TAUTE and HUBER, in 1919, inoculated themselves and 129 natives, *i.e.*, they made 131 experiments. Results were uniformly negative, and Dr. YORKE now says that more than 130 experiments would have to be conducted before one could obtain positive results. I may be unduly obtuse, but I cannot see that, in this matter, Professor YORKE's attitude is very different from that popularly known as "Heads, I win; tails, you lose!"

Dr. B. BLACKLOCK: There are one or two points I would like to ask about. Professor YORKE mentions the recommendations of the French Commission, that pigs should be used, being resistant animals, as a screen to protect native villages from attacks of glossina and from being infected. The connection of the pig with the fly has been worked out by several observers. In the Island of Principe, the Portuguese Commission said that the absence of the tsetse fly from the Southern part of the Island was due to the absence of the pig in that part. Further observations were recorded by MACFIE from Nigeria, and he quotes Dr. FORAN on the relationship between *G. tachinoides* and the pig. Pigs are known to be naturally infected with trypanosomes, as has been proved on many occasions. In Principe and the Belgian Congo, pigs were heavily infected with trypanosomes, and in Principe a cattle trypanosome of a polymorphic kind was inoculated into a pig successfully. The trypanosome appeared to be like *T. gambiense*. Inoculations have been done into pigs with animal and human trypanosomes, and in BECK's gambiense case the pig remained infective six weeks. The most interesting experiment of this kind

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is that of MESNIL and BLANCHARD, who infected one pig with *T. gambiense* and one with *T. rhodesiense*. Neither of the pigs shewed symptoms for 48 days, then they became paralysed, and after over 100 days the rhodesiense pig died. At no time did direct examination reveal trypanosomes in the blood, but mice inoculated every tenth day from their blood were infected with the disease. In the gambiense pig sub-inoculations were successful to the 60th day, and in the rhodesiense pig to the 80th day. In view of this close connection between the pig and the tsetse fly, and in view of the obvious fact that the pig can harbour various forms of trypanosome, including two of the human forms, I ask what is the underlying idea in the recommendation that these animals should be used as a screen to protect human beings, or native villages? I ask whether it is proposed to atoxylise those pigs at the same time as the humans are treated; otherwise one cannot but see disaster approaching villages surrounded by pigs.

I would refer to some statements which have been made, I think in reference to an experiment on a limited scale which has been proposed for the removal of big game from an area. The criticisms which have been directed against this proposal are somewhat various. Some critics say you cannot possibly do it, that you cannot clear completely of game any limited area. Some say it is too expensive, that the money for it cannot be obtained. Others say you may be able to get the money, but even if you clear the game away, the fly will not go. A fourth set of people say you can do all the previous things and the fly will go, but when you have banished the fly you will not be able to apply the knowledge you have got. I cannot see the logic of such statements. Why should we not apply knowledge if we have obtained knowledge? We do not know whether we can obtain the knowledge until we try experiments.

There is also another kind of objection raised to this experiment, and I am sorry to say I heard Dr. MARSHALL use these words: that there are certain hot-heads who are for the wholesale destruction of game in Africa, and I think Major AUSTEN said he was against wholesale destruction of game in Africa. For men who have heard this subject discussed for years to propagate such impressions as to the intention of those who advocate an experiment is a pity, and what they say is certainly not due to lack of intelligence. No sponsor of this experiment has suggested the killing of game in a wholesale way in Africa: that would be contrary to the very nature of an "experiment." To make such statements is to misrepresent the facts entirely, for whose benefit I do not know; it cannot be for ours, but I do not understand the reason for such misrepresentation. If we take the area involved in the experiment and the numbers involved, I suppose it might be taken as comparing the area of this room, and the number of people in it, with the area and numbers in the whole of London. It would be very deplorable if, through some misfortune, we in this room were suddenly to be exterminated, and it would, we flatter ourselves, mean the loss of important scientific men, but I do not think anybody walking down the Strand or Regent Street to-morrow morning would be any the wiser. I think that is the sort of relation this proposed experiment in Africa would bear to the whole of that Continent.

The Glossina Committee appear to have diffidence in allocating areas to the members who will work at the subject in Africa, but you will notice that they exhibit no such diffidence in being prepared to direct from London the whole of the operations of those Commissions when they are at their work, and that is a very much more courageous proposition that the mere allocation.

A suggestion which seems to me to have some importance is, that we have not, so far, done badly in the way of finding out some connection between fly and the protozoon and the disease. The work of BRUCE, DUTTON, KLEINE, KINGHORN and YORKE has not occupied a very long period, and the work has been, I think, very quickly done in comparison with that on many problems of bacteriology carried out by earlier observers, and has taken into consideration all three factors: the fly, the protozoon, and the animal or man. When such successful results have been attained by certain methods of research, I maintain we have only to increase the application of those methods of research, employing more entomologists, more men who are studying protozoology, and more men to study and treat the disease; these men should work in a state of co-operation. As

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the research will involve a large amount of experimental work, which I think it impossible to expect from isolated units of men, no matter who they are, studying in the wilderness of Africa, it is necessary to centralise in a large Commission.

Dr. A. G. BAGSHAWE: As I was another member of the much-criticised Glossina Sub-Commitee, if I were not to say something it might give rise to misconception. In fact, I came with notes, prepared "to keep my end up," but Dr. GUY MARSHALL has done that so excellently for me that I shall refrain from saying anything on that head. He has shewn that this scheme which has been critcised by Dr. YORKE is not a cut-and-dried scheme, but one which can be modified. The method of carrying it out will depend very much on the funds which can be obtained.

Professor YORKE would have the work of entomological, medical and veterinary research into the problem of trypanosomiasis combined under one central organisation in Africa. I wonder if the time is suitable for a great investigation of that kind: it would cost a great deal of money, and, if the funds were not ample, the pinch would be felt either by the entomological or pathological side; hitherto it has been the entomological side which has suffered. I doubt if it is practical politics to explore all the links of the chain of the causation of trypanosomiasis on a great scale at once. It is the entomological link which has been least explored, and it is proposed that entomologists should now have their chance. The disease has never been systematically approached from the insect side. I incline to think that the restriction of fly is at least as likely to be realised as is the restriction of game, including under that head such animals as pig. At the moment both seem to be extraordinarily difficult problems. Professor YORKE still believes that the morphologically identical trypanosomes which infect domestic animals and man respectively in East Tropical Africa are identical biologically. But, apart from the results of TAUTE'S and HUBER'S gigantic experiment, there is the epidemiological objection to accepting his view. In the late campaign in East Africa there was no case of trypanosomiasis in man met with among British or German troops north of 9° S., though tsetse-fly abounded and draught animals succumbed. It was not until the Germans got to 9° S. that they found human trypanosomiasis. Between that line and the other side of the equator there is a large amount of morsitans, and this trypanosome of game which corresponds morphologically to the human one has been found many times, and it has been found on the West Coast of Africa too. But no human case of the disease has been found north of the 9° line near the East Coast.

Now, to pass to another matter, which has not been touched on to-night. In the report of the French Committee there is a point which Professor YORKE has not mentioned – of course he had not time to mention everything. The French Committee say that in French West Africa epidemics of human trypanosomiasis are due to mosquitoes, which, in their opinion, transmit infection from one member of a family to another, leading to the so-called house or family infection. The endemicity is due to the Glossina, but the epidemics in villages are due to transmission by domestic mosquitoes. The Governors of the French Colonies in West Africa, in accordance with this advice, have enacted that all Europeans leaving French Possessions in Africa should be examined for trypanosomiasis, and, in the event of their being infected, that they shall receive an injection of atoxyl before they embark. It seems very sound for their own sakes that such cases should be discovered early, but the reason is lest these persons should infect their cabin companions through the agency of mosquitoes on the voyage home.

Infection through the agency of mosquitoes is against British experience. Obviously it might occur, but I know of no record that it has, and this French measure seems to be an ultra-precaution. In the great epidemic in Uganda, though large numbers of natives visited the lake shore beyond the narrow fly-infested zone, and returned to their homes with sleeping sickness, no case of transmission of the disease to a person who had not visited the lake shore was reported or discovered, though in many of the villages concerned mosquitoes must have abounded.

This goes against the theory that the spread of sleeping sickness in the great epidemic on the Victoria Nyanza was due to mechanical transmission. If it was due to mechanical

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transmission by *G. palpalis*, one would think there must have been a few cases of mechanical transmission through the agency of other biting flies which occur far from the lake, but this never occurred.

A better instance is afforded by the conditions in Principe and S. Thomé, which have been already referred to. In Principe, some years ago, sleeping sickness was rife, whereas in S. Thomé, a few miles away, not a single indigenous case could be found by the Portuguese investigators, though many infected persons had been imported from Angola. Both islands had abundance of biting insects. The only difference was that S. Thomé had no tsetse-flies.

Dr. G. C. Low: We have had both sides of the question—the entomological and the medical—discussed to-night, and it is evident that there is still a great amount of doubt and uncertainty as to many of the points concerned. We have heard that the French authorities propose to treat with atoxyl all infected natives in their areas, so as to destroy the trypanosomes in their blood; but will this be successful if antelopes and other game are reservoirs of infection? Can entomologists alone settle this very important point?

Dr. Guy Marshall: No.

Dr. G. C. Low: Well, that is the whole thing; for settling this and other equally important questions a combination of medical specialists, protozoologists and entomologists is required, and not one of these branches alone.

As regards Rhodesian trypanosomiasis, white people infected die with great rapidity, and some natives do so also. It is, however, possible that the disease may last longer in the latter in certain instances, and only large numbers of blood examinations can settle this. To me it looks far more likely that man gets his infection from the game, because human cases are few and far between. Again we await a definite solution of this, and until we get it money will be thrown away.

The talk of destroying the whole of the game of Africa is generally indulged in by people who have never being in that country. I would suggest to such enthusiasts that they first try to destroy the rats in a small farm at home before airing their views as to destroying the fauna of a whole continent. The only way an experiment of destroying antelopes and the larger game can be carried out is to enclose a large area with fencing so as to prevent other animals from getting in, but, even then, how can one be certain that small rodents, pigs, and animals of that sort are not present? The fly might even take to feeding on birds in lieu of game.

All these matters require careful consideration before any large scheme and spending of public money takes place. I heartily agree with Professor Yorke's suggestion that this Society should have a say in such matters. We have in it men who know the subject from every standpoint, and a discussion such as we are having now shews how useful and valuable such knowledge is.

There are many other points I should like to raise but time presses. I shall limit myself to only one more question. If we destroy these special areas on the shores of the Victoria Nyanza, mentioned by Dr. Marshall, what proof have we that the *G. palpalis* will not adapt itself to other areas? If anopheline breeding-grounds are destroyed, we know the insects will lay their eggs on water in barrels and other sites never selected under natural conditions, and the same may hold good for tsetse-flies in depositing their eggs in special soils under special conditions. The habits of *G. palpalis*, as Dr. Marshall has already mentioned, are much different on the West Coast of Africa than in Uganda, approximating more to those of *G. morsitans*. Here, again, we have another important point requiring solution. I would suggest that for further work on the subject there should be a happy combination of all experts, and that the research should not be limited to any one individual branch.

[Adjourned to Friday, November 19th.]

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Proceedings of a Special General Meeting of the Society held on Friday, October 15th, 1920, at 8.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1, Professor W. J. R. SIMPSON, C.M G., M.D., F.R.C.P. *(President)*, in the Chair.

The following Resolution, proposed by Dr. A. BALFOUR and seconded by Dr. A. G. BAGSHAWE, was put and carried *nemine contradicente*:—

"That the Annual Subscription be hereby raised from One Guinea to One and a half Guineas as from April 1st, 1921, and that the Composition Fee be raised on the same date from Fifteen Guineas to Twenty-two and a half Guineas."

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TRANSACTIONS

OF THE

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

NOVEMBER 19TH, 1920.

VOLUME XIV. No. 4.

Proceedings of a Meeting of the Society held on Friday, November 19th, 1920, at 8.45 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Professor W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P. (*President*), in the Chair.

RESEARCH INTO THE TRYPANOSOMIASIS PROBLEM: A CRITICAL CONSIDERATION OF SUGGESTED MEASURES.

BY PROFESSOR WARRINGTON YORKE, M.D.

CONTINUED DISCUSSION.

[Professor YORKE was asked to reply at this stage because Mr. DUDGEON's paper had to be taken at 8.45, and it was uncertain how much time would be available for the Trypanosomiasis Discussion.]

Professor WARRINGTON YORKE: Whilst listening to the remarks of those who took part in the discussion which followed the reading of my paper, the first thing that struck me was that, curiously enough, Dr. MARSHALL and Major AUSTEN both took exception to the fact that I had ventured to criticise the report at all. Dr. MARSHALL says I did not win from him approbation for my methods, and Major AUSTEN stated that the Report of the Glossina Sub-Committee was never published; that it was a private document, drawn up to formulate their own ideas, in order—and this is significant—that they might be able to raise funds for what they intend to do. What happened was that, in June last, I was invited to attend a meeting of entomologists from all over the world, and there I heard discussed this very report which I criticised at the last meeting. The principal speaker at that meeting took this report as his text. I took the opportunity of criticising it on that occasion, but only briefly, because the report was not before me—I simply criticised the digest which I heard at the meeting It was public property from that moment. The second point is that it is printed, and at Government expense. Finally, it is proposed to raise money on it, and to spend public funds. Now, if one cannot criticise a document which is being used to raise funds from the public, what are we coming to? Suppose the report is of the tentative nature which these gentlemen suggest, surely, now is the time for criticism, before it gets crystallised in such a way as to waste money and waste time. Or are we to consider that the remarks which emanate from this Committee are beyond criticism and above reproach? I hope not. Therefore, I think, I was justified in criticising this report.

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I was consulted about this report by the principal medical officer of one of our colonies. The report had been sent to him for comment. I think, as Dr. MAY's name has been mentioned, there is no harm in saying he consulted me, and that his views on the subject are similar to mine.

I am glad, however, that the fact that these gentlemen hold these views did not prevent them attending the meeting last month, and endeavouring to restate their position by a somewhat violent counterattack.

Let us take some of the points raised by Dr. GUY MARSHALL. He said that I objected to the Colonial Office consulting the Imperial Bureau of Entomology, and it alone, on what is a purely entomological question relating to the tsetse-fly. But it is not a purely entomological question, and never has been. Flies *per se* matter nothing, but the diseases they convey do. The point is that these flies produce disease in man and stock, so that the matter does concern us as medical men as well as the veterinarians and the entomologists. Dr. MARSHALL took exception to my remark that the report of this Glossina Sub-Committee was called for directly as the result of the report of the Earl of Desart's Committee. Well, all I can say is that I was given to understand that that was the case; and I hope it is the case, because it is lamentable if, after spending so much money and time on a gigantic Inter-departmental Committee, the Government did not take action on the report of that Committee. I do not think the Colonial Office or the Secretary of State could have done otherwise than take action as a result of it. Dr. MARSHALL says—and this is a volte-face on his part—if all the mammalia could be exterminated from a given area no one would doubt that the flies would disappear. I am glad to have had that admission from him. But that is not the only point which interests us. A great point is this: Suppose you eliminate these carriers of trypanosomes, what would be the effect on the infectivity of the flies? That is a matter which requires investigation.

The next point is hardly relevant—but many things in the criticisms were not relevant. Dr. MARSHALL quotes the work done by Mr. FISKE, who has shewn that the maintenance of the fly population in a fly area is due to the existence of a number of foci presenting optimum conditions of food, shade, and breeding-places. He says it is not therefore necessary, as has been previously supposed, to destroy all the shade in the fly area in order to get rid of the flies, which is economically impracticable, but only the shade in the vicinity of these foci; and this discovery has rendered clearing operations much more feasible as an effective sanitary measure. If that is not an example of a drowning man clinging to a straw, I do not know what is. Dr Low did well to point out the fallacy of such a generalisation on hypotheses which are far from proved.

Dr. MARSHALL says, in general criticism, that my suggestion that effort should be concentrated in one place instead of being dissipated is a result of not attempting to consider the subject from an entomological standpoint, but that I have in view merely the inauguration of a game-destruction experiment in relation to morsitans. That is not correct.

Turning now to Major AUSTEN's remarks, he complains that there is a trail of sleeping sickness over my paper. There ought to be a trail of trypanosomiasis over it. I rather admire Major AUSTEN when he goes into figures. He plunges courageously not only into figures, but into the epidemiology, and even into the pathology of the disease. You will remember he came to the last meeting armed with an enormous Blue Book, the Report of the Inter-departmental Committee on Sleeping Sickness, and read copious extracts from it. He told us how he asked Dr. MAY, in the course of his examination before that Committee, in how many instances out of the 40,000 natives in Northern Rhodesia examined for sleeping sickness, the blood was examined, and that Dr. MAY had answered about 20 per cent. Major AUSTEN went on: "In other words, while but 40,000 out of 100,000 natives in sleeping-sickness area were examined for sleeping sickness at all, it was only in the case of 8,000 individuals that an actual blood examination was made." He then asks, "Does Dr. YORKE believe that this examination of the blood of 8,000 out of 100,000 people is sufficient to prove that not one of the remaining 90,000 had trypanosomes in the blood?" It is difficult to follow Major AUSTEN's logic, but to comfort him, I can assure him that Dr. YORKE believes it no more than he believes that

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if Major AUSTEN went with a boy to Central Africa, he could solve the tsetse-fly question. How could anyone believe anything so absurd? Again, Major AUSTEN asks: "Does he (Dr. YORKE) maintain that his native, who was alive and well a year after being proved infective, was the only exception to the rule in Tropical Africa, and that he was so unfortunate as to come upon the single exception?" I never suggested anything of the sort; it is another example of Major AUSTEN's argument. What I did say was that we found one case, the case which has been quoted so often; one case who harboured trypanosomes in his blood for a long time without shewing symptoms of the disease, one case among the many found infected. Similarly, Dr. MAY stated of the 7,000 to 8,000 persons examined out of a population of 100,000 a certain proportion was found to be infected, but neither he nor any other person of ordinary intelligence believed that they were the only cases which were infected among the 100,000; he said that they were the only cases which were infected so far as we could ascertain in the 8,000 individuals, not in the whole 100,000 people. Is it likely that these odd cases of human trypanosomiasis constitute a reservoir of the human virus, comparable with the millions of antelope which are always exposed to the fly?—that is, assuming my contention is correct that the antelope harbours the human parasite. That is the question. Heaven knows what Major AUSTEN's point is.

In dealing with TAUTE's remarkable experiment, Major AUSTEN quotes a remark I made when giving evidence before the Inter-departmental Committee in, I think, 1913, that if TAUTE had inoculated a hundred human beings, the result would have been very different. Well, that computation was perhaps not justified; I had been subjected to some hours' pretty close examination by, I think, some rather hostile people, in that they did not hold my views,. And I think a hundred was not a bad estimate considering that the statement was made seven years ago, and that one had not any time to consider the matter thoroughly. If BRUCE's work, and KINGHORN's and mine is correct, then in order to determine the chances of infection one must know the number of tsetse-fly in Rhodesia, the number of times they bite the individual, and also the number of infected cases there are! Therefore I withdraw that hundred, and apologise for having, seven years ago, been induced to state it.

I was disappointed with Dr. BAGSHAWE, because he said he came with notes prepared, to support his contention, and I thought we should have something worth hearing; but he only said that Dr. GUY MARSHALL has done this so excellently that he would refrain from saying anything under that head. That was a bitter disappointment for me, because I had not considered Dr. MARSHALL's remarks in that light. But Dr. BAGSHAWE does make one, and as far as I can make out, the only real, relevant point in the discussion, and that is the epidemiological point, where he points out that the Germans did not encounter sleeping sickness north of the line 9° S. I cannot answer that point at present, but I am sure Dr. BAGSHAWE will be the first to acknowledge that a great deal more knowledge is required on the epidemiology of Rhodesian sleeping sickness before we can make a deduction from that.

Dr. BAGSHAWE also referred to Dr. DUKE's remarkable work, which resulted in his striking theory of mechanical transmission, but that is outside the scope of this paper, and so I do not propose to discuss it.

I submit that the members of this Glossina Sub-Committee have not answered my criticisms of their report. My criticism is two-fold—and one has to repeat it time and time again—that the problem is not a purely entomological one; it is the disease in man and in stock which matters, not that there are so many millions of flies in Africa. They are annoying, but what we bear them a grudge for is the disease which they carry. I hold that previous work—not only that of KINGHORN and myself, but that of the Royal Society's Commission, work which called forth that Committee—indicates that the further line of research should be in the direction of ascertaining the relationship which exists between the game and fly and the trypanosomes of man and of domestic stock. That has not been touched upon.

My second criticism is that the organisation, which this Glossina Sub-Committee proposes to set up to investigate the problems which it itself states should be solved, is

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not competent to do anything. It will fai lamentably. It is no advance on what has been existing in Africa for the last ten years They propose to send to Africa twelve entomologists—if they can get them, and it is admitted that they are not available at the present time—to send them to six different places, thousands of miles apart, and to leave them there, with the sole stipulation they should report to London, to the Glossina Sub-Committee. These twelve isolated entomologists will be expected to answer questions which can only be solved by a gigantic organisation, involving a great outlay, and a concentration of energies.

Those, in brief, are my criticisms of the report of this Glossina Sub-Committee, and I think those who read the replies to those criticisms will realise, as I realised, that the replies evade the point, and put into my mouth, as was done seven years ago, statements to which I never gave utterance, and against which, of course, I protest.

Sir JOHN ROSE BRADFORD, F.R.S.: As a member of the Departmental Committee which has been alluded to, I would like to make a few remarks.

I think the Society is to be congratulated on this paper by Professor YORKE. Unfortunately, I was not here last time, but, though I had not the opportunity of hearing it, I have read it, and there is no doubt it is an interesting paper. It is also interesting to the Society, because, whatever its other merits may be, it certainly, as I gather from what has passed to-night and from what I have read, led to a very lively discussion.

I do not propose to deal with any of the special technical or scientific questions, because my first-hand knowledge of trypanosomiasis is limited, as it is some years since I worked at this subject and I am not thoroughly familiar with the subject now. So in the few remarks I make I would like to deal with the matter from a general point of view.

One of the things which struck me in Professor YORKE's paper was his criticism that the Government had not approached this Society in the matter, and he made somewhat unfavourable comparisons between the methods of the British Government and the methods of the French Government. I think that that criticism, in this particular instance, was a little beside the mark, if I might say so, because although, doubtless, the Government might derive great advantage from consulting this or other societies in matters of scientific moment, the comparison with the method of the French Government was not quite apt, because the Government did not consult, so far as I understand, the Glossina Sub-Committee on this proposal. It is not a question of the Glossina Sub-Committee *versus* this Society, but the real comparison with the French is the comparison between consulting a Society, and appointing a Departmental Committee. The Departmental Committee appointed some years ago was the step taken by the British Government, which is comparable to the action of the French Government in consulting their Tropical Society. That Departmental Committee, whatever its faults may have been, was a very remarkable Committee, because it was a Committee with such wide interests. I have served on many committees in my time, but I doubt whether I have served on one of such striking character as that one, owing to the very varied interests which were represented on it. I do not think any society could have included amongst its members representatives of such varied interests as that Committee contained: sportsmen, naturalists, entomologists, pathologists, physicians, officers with administrative experience. Government officials; it was a very large and representative Committee. It sat for a very long time, and took a great deal of evidence on a very difficult problem. So far as my memory serves me, that Departmental Committee was originally constituted owing to the great conflict in opinion amongst scientific men and amongst naturalists and sportsmen, with reference to the question at issue, *i.e.*, the relation of game to sleeping sickness, and allied diseases, and especially with reference to the destruction of game. Anybody who served on that Committee, or anybody who read the evidence which was brought before it, must realise the extreme difficulty and complexity of the subject. That is seen in the report issued. I think if any comparison between the action of the British Government and the action of the French Government is made, the real question is whether it is better to appoint a Departmental Committee or to consult a Society. That comparison in the present instance, however, does not arise, since the real question is

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this: Since that Committee met has there been any advance in scientific knowledge which completely alters the point of view from what it was at that time? I confess I am not thoroughly familiar with the matter now, but I am not aware that in the last few years there has been any revolutionary advance which puts the subject in a totally different position from that which it assumed at that time. Therefore I do not think there is very much in Professor YORKE's criticism as regards the action of the Government.

To go back to the Departmental Committee. That Committee recognised, as Professor YORKE points out, and I think everybody in this room will agree, that the problem is a mixed one, partly entomological, partly pathological, and so forth. The Committee recognised that, and it was brought forcibly before that Committee that, although much scientific work had been done in the last few years on the purely pathological side, no corresponding amount of work has been done on the entomological side. The members of your Society and the like are those best qualified amongst pathologists, perhaps, to realise the great services which entomology has rendered to pathology and to medicine. It does not seem to me a very strange act on the part of that Departmental Committee to advocate in their report further entomological enquiry. Furthermore, I think I am right in saying there is more obscurity about some of the entomological problems connected with Glossina than there is in regard to some of the more pressing pathological problems. So that there was much to be said for an extensive entomological enquiry. The summary of the report—the practical result of that Departmental Committee—was, to my mind, two-fold; firstly, they recommended a game-destruction experiment, with certain limitations. They were bound to point out the extreme difficulties of carrying out such an experiment; everybody must admit that. It is a very desirable thing to do, but it is open to many fallacies, and it is one which would be extremely expensive. Secondly, they reported that it was very desirable to prosecute entomological enquiry. Looking at it now from the point of view of an outsider, I say that, although, no doubt, it is a counsel of perfection to have an enquiry and an organisation which will deal with the whole problem—pathological, clinical, entomological and so forth—yet that is no reason why impediments should be raised or difficulties placed in the way of carrying out a portion of the enquiry, namely, the entomological portion, which must, humanly speaking, bring forth results of some value, and in all probability results of very great value. You all know, in medicine, instances where the spread of certain diseases has been due to insects, and where the prevention of such disease has been carried out by methods derived from entomological enquiry, and where knowledge concerning the disease, and of the virus causing the disease, is in a far more chaotic condition than it is in the case of trypanosomiasis. So it seems to me it would be highly desirable for an entomological enquiry, presumably on the lines recommended by the experts of the Glossina Sub-Committee, to take place; and it would be a pity for impediments to be put in the way of that, simply because it does not deal with the whole problem of trypanosomiasis.

Dr. A. S. NEAVE: I also would like to thank Professor YORKE for providing us with a paper which has given rise to an extremely interesting discussion, even though I confess I shall differ from him fundamentally.

Before I state my own views on the subject, I would like to refer to the question which he himself has just raised as to publication. Nothing that Professor YORKE has said this evening seems to alter the fact that the report he has been criticising has never been published. It was discussed, it is true, in an indirect manner, at the Imperial Conference, because the principal speaker was a representative of one of the Governments to which the report had been sent with the intention of getting their views, and, on his own authority, he used some of the data in that report for the paper he read. Nevertheless the fact remains that the report never has been published. I think Professor YORKE is wrong in supposing that the speakers who oppose him object to his criticism of the report as such. The point is—I say it with diffidence as a visitor—ought the criticisms of the report in question to be made more public that the report itself? Professor YORKE's criticisms

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are most valuable, but it is one thing to criticise such a report in this room, and another thing to publish such comments on an unpublished document.

Now with regard to the report itself, Dr. MARSHALL is said to have pointed out—and I think Professor YORKE has treated Dr. MARSHALL's speech rather unfairly, by extracting one or two isolated sentences and divorcing them from their context—that the *question* was a purely entomological one. This is not the case, and no one, I imagine, thinks it is. The *report* of the Glossina Sub-Committee is a purely entomological one, and as Dr. MARSHALL has said, the remarks Professor YORKE made on the relation of the Glossina Sub-Committee to Lord Desart's enquiry were based on a misconception. But let us suppose he was right; surely the result of Professor YORKE's conclusions would become all the more surprising. As has been pointed out by others, and is also quoted by Professor YORKE himself, the Desart Committee attached great importance in its report to a proper scientific equipment of entomological research. If so, was it surprising that a purely entomological Sub-Committee, appointed by an entomological body such as the Imperial Bureau, dealt solely with the entomological side of the question? It seems to be precisely what we should have expected.

We have here a problem in which we desire to exterminate from the world a blood parasite with a vertebrate host and an insect vector. There are consequently two main lines of investigation. That is to say, we must endeavour to discover methods of exterminating either the vertebrate host or the insects concerned. Of the two, it seems to me that attempts to destroy the insects are more likely to meet with some measure of success. In the first place, they are less likely to interfere with the balance of nature in Africa, and that is a factor of vital importance. Half mankind's difficulties in dealing with insects, whether disease-carriers or plant pests, and with practically all the higher organisms, have been due to the upsetting of the balance of nature. And that is a point we should bear in mind. Secondly, attempts to destroy the fly are less likely to interfere with the convenience of the human inhabitants of Africa than is the destruction of vertebrates. I think our chances in succeeding in dealing with the fly are better than those in trying to exterminate such vertebrates as may be proved to be its hosts.

The holding of that opinion does not denote any objection to carrying out experiments on protozoological lines, such as Professor YORKE wishes to see. Professor YORKE, on the other hand, wishes to confine investigations to experiments on the destruction of the vertebrate hosts, while at the same time putting difficulties in the way of entomological research. A small point in regard to this was raised by Dr. BLACKLOCK at the last meeting, and is worth examination. He took to task several speakers because they had talked about total extermination of game, and said that such a thing was not suggested, nothing more than an experiment being contemplated. The logic of that position is difficult to comprehend. Is Dr. BLACKLOCK only prepared to advocate experiments on game destruction without carrying them to a logical conclusion? It seems to me that the opponents of game-destruction experiments base their objections not on the fear that all game may have to be destroyed, but on the fact that we do not know enough about the subject at the present time to devise a means of carrying out such experiments on lines the results of which will have general acceptance. The fear is that there will only be an expenditure of money without getting a result that we can accept. I think a game-destruction experiment may be justified in years to come, but at present I do not think we can devise conditions under which we can secure a satisfactory result, one which will appeal to the scientific mind. There is the great difficulty of having a control under the conditions of the experiment, How can you arrive at a result in any game-destruction experiment over a given area unless you study carefully and over long periods the conditions in the area before and after the experiment is made? Professor YORKE himself sees this difficulty, and in his paper called attention to the risk of that point of view being overlooked in the experiments being conducted in Zululand. The fact of the matter is, that in the present state of our information a game-destruction experiment would be of doubtful value, compared to a deliberate attack on the fly based upon a study of its habits. We do not know enough about the species of Glossina such as *G. morsitans*: if we knew as much about them as we know about *G. palpalis*, the situation would be a

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good deal clearer, and that information might lead us to hold opinions very different to what we hold at present as to the nature of the fly's dependence on game. It is clear, from FISKE's work, that to exterminate the main food supplies of *G. palpalis* on the Victoria Nyanza, you would have have to kill off, not the antelopes, but the lizards and crocodiles. I ask you, How would you propose to set about this on the Victoria Nyanza? When we come to *G. morsitans* and *G. pallidipes*, we will find that the favourite host is some such animal as the bush pig, an animal that would be excessively difficult to exterminate in any part of Africa.

It seems to me that there is only one way, so far as we know at present, of exterminating Glossina, in the sense of rendering an area permanently free from it. That method is the occupation of the area and the country generally by man on a large scale; under such circumstances the destruction of game would take place side by side with that of Glossina. I do not think that at the present moment any form of game destruction, nor even any form of fly destruction, would have more than a temporary effect in driving the fly back. But I think we have some hope of a permanent result if entomological investigations are carried out on the lines recommended by the Sub-Committee, and if our workers would supply evidence and knowledge as to *G. morsitans*, such as FISKE has produced in regard to *G. palpalis* in Uganda. By a combination of various methods—driving back the vertebrate hosts, partial clearing, introducing parasites and traps in the breeding places, and so on—we might render an area habitable to man and his domestic animals at the moment we render it free from fly, always provided that man was prepared to occupy the area immediately. I think the extermination of the fly can be rendered largely automatic, provided man is living sufficiently densely in or near the area it is required to clear.

Professor YORKE objects to the scope of the scheme drawn up by the Glossina Sub-Committee, and thinks it represents no advance on the bad old days of the last ten years or so. Surely, he is somewhat mistaken. The recommendations of the Sub-Committee go a long way beyond the days when it was thought sufficient to attach to a Sleeping Sickness Commission one medical officer, who was supposed to have some knowledge of entomology. The Sub-Committee recommend that there shall be a *minimum* (not a *maximum*, as Professor YORKE suggests) of two entomologists working together at one time, whereas the few entomologists engaged up till now on this subject have had to work singly. I admit it is difficult to get the trained men we want, but is that our fault? We can put our hands on five good men, who know the African conditions. It must take time to produce more.

I find myself bound to disagree profoundly with Professor YORKE in regard to his general conclusions. And I cannot quite understand his basic objection to carrying out entomological research, nor why he criticises such work. It is true that it is not the line of work he is particularly interested in, but it has the same object, and hence should have credit therefor. Surely, he might leave it to the entomologists to work out their line, as he works out his.

Professor R. T. LEIPER: One point which has struck me very forcibly, in listening to this discussion, is the very large sum of money which it is proposed to ask the Colonial Office to devote to this particular line of enquiry. It has been suggested that jealousy on the part of Professor YORKE has given rise to his protest, but is it not his longsightedness? I am sure he sees that if the Colonies are to be asked to spend £50,000 *at least* on the purely entomological side of this investigation, there will be very little money available from these Colonies during the next five or ten years for cognate researches in trypanosomiasis or any other subject of importance in Tropical Medicine.

It should be recalled that the Colonial Office receives in small grants of £100 or £500 from each Colony a total sum of perhaps not more than £10,000 a year for the service of its Scientific Committees and their publications, and that the cost of the enormous research organisation built up during the war in this country by the Medical Research Committee came to less than £40,000 per annum.

The time has come when some one should make the suggestion (and I venture myself

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to commit the indiscretion) that the Colonial Office would be well advised to co-ordinate the work of its various bodies and committees like the Tropical Diseases Research Fund, Tropical Diseases Bureau, and the Imperial Bureau of Entomology by appointing a Tropical Research Council on lines similar to the admirable Medical Research Council which has recently been formed for the co-ordination of research work under Government auspices in this country. Increased support from the Treasury might be forthcoming if a close link were established with this Council.

It would be the duty of the Tropical Research Council to enquire into the needs and to delimit the activities of the various bodies receiving Government aid.

Colonel D. HARVEY: I would like to make a few remarks on the discussion, but I have no intention of entering into the controversy.

Professor YORKE, in his paper, referred to the fact that he had found 16 per cent. of the game in the district where they were working, the Luangwa Valley, were infected with trypanosomiasis, with the organism pathogenic to man, and he said that Commission, of which I was a member, confirmed this fact. That is hardly how we should have expressed it. I am giving my own ideas, as Sir DAVID BRUCE is, unfortunately, away from England. If Professor YORKE had said we found that the trypanosome that was present in cases of human trypanosomiasis in our district of Nyasaland was identical with the trypanosome which we found in game, I should have been at one with him. But when he says this trypanosome which they and we found was pathogenic to man, that was just the bit of evidence that was wanting. We spent three years trying very hard to identify as one or separate these two trypanosomes, but at the end of that time we had to confess we had not done so. The trypanosomes of game and those in man were the same morphologically in their action on experimental animals and their development in the fly, yet we could not say that the game trypanosome was pathogenic to man. TAUTE came to our camp, and stayed a fortnight, and we said to him, "All that remains now is to find out whether this game trypanosome is pathogenic to man." He said, "I am convinced it is not," and he added, "I am willing to demonstrate it on myself." We said, "Very well, we have a nice strain of game trypanosome here." He replied, "I will not do it here; you have the human trypanosome in your district; I will go to the other side of the lake, where there is no human sickness, and I will carry out the experiment there." He went off, and shortly afterwards we received a letter from him, giving full details of the experiment on himself, shewing that he, at least, had not been infected either by the fly or the inoculation of the blood of infected animals. We said that this shewed only that TAUTE himself was immune to this particular trypanosome. Therefore, that point had not been settled. The only other thing we could see that remained to be done was to work out carefully the epidemiology, that is, to find out whether this disease was widespread wherever the game was to be found. We had to leave the question like that because we could not say more. Sir DAVID BRUCE stated in the report, "It will be found difficult, or even impossible, to arrive at a certain decision with regard to identifying these two strains."

The only fresh evidence which Professor YORKE tells us about are these further experiments of TAUTE's, and Professor YORKE says he does not think this carries us much further. But I think it carries us to this point, that the strain of trypanosome which TAUTE was working on was not pathogenic to man.

The only other piece of evidence which has come along since is the epidemiological evidence which was afforded during the war, and which Dr. BAGSHAWE referred to. We have had interesting reports about that. Not only German but our own troops—South African and British—went through German East Africa, and it was only in certain parts—although morsitans is widely spread all over the country—that a few men were affected with the Rhodesian sickness. These regions were known before as the parts where this sickness was to be found, and it was when the troops got down near the Rovuma River, and one main road in Portuguese East Africa, that one or two men were infected. When the men went to a certain part of this road, half a dozen of them contracted rhodesiense sickness. This evidence, therefore, favours the view that the human Rhodesian trypanosome is a different strain, though a similar trypanosome.

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Professor YORKE said the only explanation he could give of the distribution of the disease was that man was relatively immune to this trypanosome. In the district where we were one would think that, if man was immune to the trypanosome, we should have found various types of severity in cases of the disease. But it was not so. When a man became infected he went down in three or four months; there were no gradings of severity in the disease.

With regard to the question of entomologists being employed for the investigation, I am at one with Dr. YORKE in saying that the way to attack the disease is to do so from all sides, and I am sure one of the advantages which Professor YORKE's Commission had in Rhodesia was that they had as entomologist with them LL. LLOYD, who did such excellent work.

Professor YORKE: May I ask Colonel HARVEY one question? He made a statement which is new to me, and which is of the utmost importance. Do I understand him to say that TAUTE, when he called at the Nyasaland laboratory and was asked whether he would be willing to inoculate himself with the strain of game trypanosome with which they were concerned, replied that he would not do this because he felt sure the human trypanosome was in that district and must, therefore, be in the game?

Colonel HARVEY: Yes, and that was also our opinion, that if you got human trypanosomiasis, the fly is infected with the parasite, and, therefore, the game of the district was also bound to be.

Dr. A. BALFOUR: As a member of Lord Desart's Committee I may perhaps be permitted to contribute to the discussion. As regards the main point under consideration I have nothing to say, but I think it is useful to speculate a little upon those problems of immunity which Professor YORKE has raised.

In the case of *Trypanosoma gambiense*, is there really a *natural* resistance of man to the infection? May it not rather be that man is the original mammalian host of this trypanosome? I confess that, along with others, I had always thought the comparative immunity of the West Coast native to be due to the fact that sleeping sickness was an old disease on the West Coast, and the native population had, as a result, acquired immunity, presumably in the same way as is seen in the case of other communicable diseases.

Again, what really occurred in Uganda? The ordinary view is that Stanley opened up Central Africa, that the West Coast sleeping sickness was introduced into Uganda, and that *T. gambiense* attacking an immune population played havoc with it, just as measles did in the case of the Fijians.

On the other hand, as Dr. DUKE has shewn, there is evidence that sleeping sickness was known in Uganda long before the outbreak of 1901. The natives had a name for it, *mongota*, signifying "to nod the head."

Was this original Uganda sickness *T. gambiense* infection?

Is it the case that merely a new strain of *T. gambiense* was introduced into Uganda? Could a new strain, apart from a new trypanosome, produce the terrible effects which occurred, and which used to be attributed to the new disease?

Is Dr. DUKE's interesting and ingenious hypothesis, cited by Professor YORKE, correct? There are, as Dr. BAGSHAWE has stated, points against it. For example, if the transmission was mechanical, why was the disease completely checked when the natives were removed from a palpalis area? Though no longer in danger from tsetse-flies, they were still liable to attack by mosquitoes and other biting insects, and the work of HECKENROTH and BLANCHARD has shewn that experimentally, at least, mechanical transmission by mosquitoes can occur.

The immunity problem in Rhodesia and Nyasaland is specially interesting. There is little evidence to shew that the disease is an old one in these regions. I remember Sir JOHN KIRK—an historic figure—who gave evidence before the Desart Committee, saying that when he was in those parts of Africa with Livingstone there was no word of any form of sleeping sickness. Are we then witnessing, as has been suggested, the gradual adapta-

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tion of an animal trypanosome to life in man, the latter having hitherto been naturally resistant to the infection, as he is in the case of *T. congolense*, *vivax*, *evansi*, *pecorum*, etc.?

If so, and if man becomes fully adapted to *T. rhodesiense*, may we one day expect an outburst of Rhodesian sleeping sickness in epidemic form? In any case it is curious that, at the present moment, *T. rhodesiense* is known to be more virulent to man than *T. gambiense*. Why then is the infection not more extensively spread? There are two possible explanations:—

1. It kills off its human host more rapidly, and hence the period of infectivity of the latter is shorter. At the same time it must be remembered that those infected live for a considerable period.

2. It may be necessary to distinguish between what we may call a pathogenic virulence and a virulence of propagation. Something of this kind is seen in certain outbreaks of small-pox, where you may have a severe but limited outbreak, and, on the other hand, a mild epidemic extensively widespread. In the case of sleeping sickness, however, the question of the tsetse-fly, of course, complicates the problem; hence the necessity for entomological research.

All these questions are at present of a hypothetical nature, but it would be interesting to hear Professor YORKE's views regarding them, although, I fear, it would take more time than we can spare to discuss them adequately.

Dr. C. M. WENYON: Dr. BALFOUR has told us that Sir JOHN KIRK had informed the Committee that sleeping sickness had not been recognised in Rhodesia, and on that account it is possible the Rhodesian infection was a new disease. Is it not a misnomer to talk about the Rhodesian disease as sleeping sickness? It does not produce sleeping sickness. A person who gets the Rhodesian sickness dies quickly of infection which does not take the sleeping sickness form. Therefore, the disease was probably overlooked.

Dr. H. S. STANNUS: I have seen trypanosomiasis due to rhodesiense infection with symptoms which could not be distinguished from those of the Uganda illness due to gambiense.

Professor W. YORKE (in further reply): Sir JOHN BRADFORD misunderstood me, for he was under the impression I was blaming the Government for not consulting this Society in this matter. On the contrary, I was rather suggesting that I thought this Society ought to be in a position—and ought to so organise itself that it should be able—to give advice to the Government on matters of the sort; that I still think is an important function of such a body as this. If we are not ready to offer advice, we are never likely to be asked for it.

Sir JOHN BRADFORD referred, at considerable length, to the Departmental Committee. The report of the Committee is published, and anyone can see what was said about the proposed game-destruction experiment. I do not wish to go into that, but I do wish to emphasise this fact most definitely: I am not placing difficulties in the way of entomological research; I want as much of it to be done as possible; I wish to encourage it. What I object to, in the first place, is the method adopted. There is an atmosphere of secrecy about it. It has even been contended again to-night that the report of the Glossina Sub-Committee is a secret document. It has been discussed publicly; certainly the principal speaker at the meeting I have alluded to discussed it. It is being used to obtain money, and not a small sum—£50,000 to begin with. This Society must feel that those responsible for that report ought to be in a position to justify that expenditure of money. I say their organisation is defective. You cannot, by planting two men—even though those two be Major AUSTEN and Dr. MARSHALL—in the middle of Africa, with insufficient funds at their disposal, without other European help, solve those problems which it is stated in the report have to be solved. That is my main indictment, and no one has attempted to reply to it.

I was very much interested in what Colonel HARVEY said; I think the question

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between us is one of terms. When I say that the two trypanosomes are not only indistinguishable but identical, I may, of course, be wrong, but that is my view. I was much impressed by what he said in regard to Dr. TAUTE, because that seems to cut the ground from under the feet of those who deny the identity of the game and human trypanosome. We are faced with this question: If TAUTE admits that antelope can be infected, and fly can infect one antelope from another, where does it end? We know flies can be infected from antelope, and flies so infected can infect other antelope. Surely, owing to the intimate association between game and fly, the process, once started, must result in the whole of the antelope being infected.

I cannot now enter into the interesting question of immunity, which was raised by Dr. BALFOUR; it would take too long to argue. We must have more work on these lines too before we can formulate an opinion worth discussing.

The PRESIDENT: I expressed my views on this paper at the last meeting, and notwithstanding the controversial nature of the subject, I still agree with Professor YORKE—as I gather others also do—that more investigation is needed. And I agree with him that it cannot be carried out by entomologists alone, but that some team-work is required. It is better, from this point of view, that effort should be concentrated on one place rather than by a number of isolated places with too small a staff in each, in the way which was proposed by the Glossina Sub-Committee. I think this debate may, perhaps, help to modify the scheme, which has not altogether been completed.

In view of the references in Professor WARRINGTON YORKE'S paper to the Report of the Glossina Sub-Committee of the Imperial Bureau of Entomology, it seemed desirable to secure permission, which has been granted, to reproduce the Report here in full, as it has not previously been published.

IMPERIAL BUREAU OF ENTOMOLOGY.

REPORT OF GLOSSINA SUB-COMMITTEE.

1. Some idea of the importance of the tsetse-flies of the *Glossina morsitans* group as an obstacle to the development of Tropical Africa may be gathered from a consideration of the wide areas over which they range. In Southern Rhodesia, in which the fly-belts are comparatively small and have been more carefully surveyed than elsewhere, their extent has recently been estimated at 9,000 square miles. With reference to N.E. Rhodesia, Messrs. KINGHORN and MONTGOMERY have expressed the opinion that it would be difficult to find a continuous area of fifty square miles free from *G. morsitans* anywhere except on the Serenje plateau, around Fort Jameson, and on the high plateau between Lakes Nyasa and Tanganyika. In Nyasaland probably more than a fifth of the area is infested by tsetse-flies; while in Tanganyika Territory the fly-belts must cover nearly half the country. No precise information is yet available as to the extent of their range in British East Africa, Uganda, the Sudan, and the interior of West Africa.

2. Before any practical measures can be devised for the successful control of these dangerous insects, it is essential that we should know a great deal more than we do at present of their habits and life histories, and of the factors which favour their increase or inhibit their spread.

3. With regard to the habits of *G. pallidipes* and *G. longipalpis* we know practically nothing at present; but thanks to the work of various observers in Rhodesia and Nyasaland, a certain amount of useful information has been acquired with regard to the behaviour, breeding places, and parasites of *G. morsitans*; yet there still remain many important gaps in our knowledge.

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4. The principal measures that have hitherto been recommended for the eradication of tsetse-flies are:—(1) the wholesale destruction of the larger mammals; (2) the clearing of all small trees and undergrowth; (3) the continuous capture and destruction of the flies on a large scale; (4) the eradication of all natural breeding places, and the substitution of artificial ones, from which the pupæ would be regularly collected, the flies emerging from them being killed and any parasites released; (5) the artificial breeding of parasites on a large scale, and the interchange of parasites from different parts of Africa.

5. Apart from various other inherent difficulties, all these proposals have one drawback in common, namely, that it is impracticable to carry them out effectively over large stretches of fly country. Nor are they likely to be of any real use in restricted portions of such areas, unless they are persistently maintained over a considerable period of time; for a relaxation of effort would almost certainly lead to a gradual re-infestation of the area. Thus, in the present state of our knowledge, it would seem that the adoption of such measures would be of little avail except where the cleared localities could at once be placed under beneficial occupation by settlers, who would maintain the disturbing factors that are inimical to the fly.

6. In the case of small and completely isolated fly-belts a combination of some or all of the foregoing methods of attack would probably be ultimately effective, if we may judge from the successful extermination of *G. palpalis* in the island of Principe, in the Gulf of Guinea. But fly-belts of this character are very few and far between.

7. A more intimate knowledge of the bionomics of these insects will probably reveal the weak points at which they can be most effectively attacked; and these are more likely to be discovered, and with less loss of time, if a comprehensive series of observations and experiments can be carried out simultaneously in various localities throughout their range in which differing conditions of life are likely to occur.

8. One of the primary requisites will be a thorough survey in each of these localities of the areas frequented by the different species; and these surveys will need to be checked at intervals throughout the year, in order to secure more exact information as to the seasonal and secular movements of the flies. In the course of these surveys the density of the fly should be estimated at frequent intervals on a standard basis, such as that adopted by Mr. FISKE and Dr. CARPENTER for *G. palpalis*.

SUGGESTIONS FOR OBSERVATIONS AND EXPERIMENTS.

Distribution of the Fly.

What are the precise factors that determine the limits of a fly area?

Is there a marked contraction of the range of the fly during the height of the dry season?

If so, what are the special features of the localities in which the flies concentrate?

If a sufficiently small focus of this kind can be found, endeavour to exterminate the flies in it by every means and render it uninhabitable for them by clearing, burning, etc.; then carefully note the subsequent effects of these operations.

If the fly is absent from places that appear to be suitable and in which food is plentiful, endeavour to ascertain what are the adverse factors.

Effects of Clearing.

Does clearing actually kill the flies or merely drive them away?

In the open "orchard bush" so much frequented by *G. morsitans* is it sufficient to clear away shrubs and branches of trees to a height of twelve or fifteen feet, or must trees of twenty to thirty feet be felled?

What is the minimum width of cleared belt that would effectively check the spread of the fly?

Study the best methods of maintaining the clearings, by the planting of crops, by burning, etc.

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Can the numbers of the fly be materially reduced by judicious burning of grass at times when food is scarce and shelter limited?

Compare the results obtained by indiscriminate clearing and by the clearing of breeding places after these have been defined.

What is the minimal degree of clearing which will render such a breeding place useless to the fly?

Breeding Grounds.

Make a detailed study of breeding places, noting their relation to soil, shade, surrounding vegetation, aspect, presence of food, proximity of water, etc.

Ascertain whether collections of puparia placed in unusual situations are more liable to destruction than in natural breeding places.

If so, what are the enemies that take advantage of the alteration?

What are the factors that safeguard the normal breeding grounds?

Test the effect of destroying natural breeding places and establishing artificial ones from which the puparia are regularly removed.

Can the attractiveness of an artificial breeding place be enhanced by means of special odours, etc.?

Ascertain the precise relation of breeding to season by the periodical examination of selected areas.

Food of the Fly.

Test the food preferences of the various species of Glossina.

Are any of them absolutely dependent on any special species of mammals?

Is there a critical breeding period during which a marked reduction in the food supply would seriously check reproduction.

Do the flies seek their food by sight or scent? and does the presence of long grass prevent them from finding their hosts?

Destruction of Adults.

Test various methods of killing and trapping the adult flies on a large scale for a prolonged period—not less than a year.

Endeavour to ascertain how long the flies live in nature by releasing large numbers of marked individuals and re-catching them at intervals.

Parasites.

Try whether it is possible to breed local parasites in very large numbers, using the puparia of common and nearly related flies that can be easily reared in the laboratory.

Test the possibility of introducing additional parasites of different kinds from remote parts of Africa.

Enemies of the Fly.

Ascertain what natural enemies there are of the fly at different stages in its growth. Are birds of any practical importance as enemies of the fly?

Influence of Odours.

Test various substances that may serve to attract flies for the purpose of trapping.

Search for an effective and lasting repellent.

Endeavour to find a substance the odour of which will stimulate larviposition.

For the elucidation of the foregoing problems it seems desirable that there should be at least six different experimental stations, widely distributed over Tropical Africa, and the countries that suggest themselves as being most suitable for the purposes are:—Zululand, Southern Rhodesia, Nyasaland, Tanganyika Territory, the Sudan and Northern Nigeria.

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The selection of the precise areas in these countries that would afford the best facilities for these investigations would require a more intimate knowledge of local conditions than the Sub-Committee at present possesses. But it may be laid down generally that the various areas should be representative of different types of environment, and that they should, as far as possible, be situated in localities in which the presence of tsetse-flies is actually obstructing settlement or is likely to do so in the near future.

Each of these stations should be under the control of a competent entomologist, and he should have at least one assistant who would be able to carry on the work in the event of the senior man falling sick or going on leave. Unfortunately at the present time there are very few men who combine the requisite qualifications and experience for this class of work. This difficulty could be met by starting in the first instance, say, two stations only. In these new men could be concentrated for training purposes. After a time the most promising pupil could be left in charge, and the others would proceed to open up a new station elsewhere.

While the observers in each station would be expected to keep an alert watch on all aspects of this complex problem, it would probably be advisable for some of the stations to give special attention to certain lines of work, such as, control by clearing, artificial breeding places, rearing of parasites, etc.

Each station ought to send in a monthly progress report to the Imperial Bureau of Entomology, so that the Glossina Sub-Committee may be kept advised of all the latest developments. They would thus be in a position to co-ordinate the work, to prevent overlapping, and to arrange promptly for the testing under divers conditions of any new hypothesis that may be put forward.

As the work progresses it is probable that special lines of enquiry will become necessary for which the aid of a protozoologist or veterinarian may be required, and provision should be made for such eventualities.

May, 1920.

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SOME IMPORTANT TROPICAL FRUIT-FOODS.

BY

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The determination of the nutrient values of different kinds of human food has formed the subject of much careful investigation during the last half century, and by analytical means it has been made possible to ascertain the correct proportion of each kind required to form combinations for sustaining human life in a healthy condition. These investigations have perhaps acquired an enhanced importance during the last few years, when the world's trade and transport was thrown into sudden disorder by the war. The immediate effect of this was to cause a cessation of the supply to ourselves of certain food commodities that had come to be recognised as essential, and to induce a search for substitutes from among the very limited material available. A less urgent, but nevertheless interesting, matter which seems to have been brought into prominence by the same cause, is that, in certain parts of the world, people have been able to find sufficient nourishment in a diet composed almost entirely of fruit and other vegetable products. In some instances it seems evident that, not only has life been sustained for long periods upon such a diet, but that those accustomed to it have been able to produce a physically strong and active type of mankind, although nourished in a manner that appeared somewhat inconsistent with that generally supposed to be necessary for such human development. In consequence, a more exhaustive enquiry into the nutrient values of their staple diets has appeared necessary.

I hasten to add, that it is not my object to attempt to solve this question or to challenge the conclusions already arrived at by scientific investigators, with respect to the formula laid down for a complete human food. It is, indeed, with great diffidence that I approach the subject of food values, and my only purpose is to furnish a little evidence, based on personal observation and experience in the tropics, supplemented by the published records of others, regarding certain fruit foods habitually used by some of the indigenous people and forming their staple subsistence.

Selections have been made of instances where it appears that some well-known tropical fruit has formed the almost entire diet. It must be admitted that in order to obtain definite and incontestable evidence of the original food of an independent race, it is necessary for observations and records to have been made before its tastes and habits had begun to be altered by association with civilisation and the accompanying introduction of new foods.

The articles of diet considered desirable by colonists and explorers follow them rapidly in their travels, and their tastes in the matter of food and drink are perhaps more readily imitated by the uncivilised races they meet than any other new custom. It is, therefore, difficult to point to the existence, now, of uncontaminated examples. Thus, in discussing the original staple diet of a race, the inference must be supported by circumstances which eliminate the probability of it obtaining much food from other sources, as, for instance, its permanently hostile relations with its neighbours and the conditions of the country of its inhabitance. A case calling for the aid of such deductions is that employed here to exemplify the use of the plantain as a staple food of the Ashanti race of West Africa.

The banana and plantain (the latter generally recognised by botanists as a sub-species)

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are selected as occupying the first place among fruits, constituting an almost complete food for some of the world's inhabitants.

Several species of plants belonging to the genus Musa (sub-genus *Eumusa*), yielding fruit with a fleshy pulp, and known widely as bananas and plantains, are extensively used as human food in the tropics. Perhaps the most important of these, of which varieties are commonly met with in cultivation, are: *M. sapientum* (and its sub-species *paradisiaca*, the plantain), *M. acuminata*, *M. corniculata*, *M. cavendishii*, *M. discolor*, and *M. malaccensis*. The first-named has so extensively spread from its original habitat in the Indo-Malayan region, that it is now found in all parts of the tropics of the Old and New World. To such an extent has it accommodated itself to its surroundings, that it has come to be regarded as indigenous in some countries where the history of its introduction is now lost. According to Sir HARRY JOHNSTON, the plantain (*M. paradisiaca*), a sub-species characterised chiefly by the comparatively small proportion of sugar contained in its fruit pulp, was first introduced and cultivated on African soil by the ancient Egyptians, and reached the negro by gradual descent from Egypt. There are several African species of Musa belonging to the sub genus *Physocaulis*, bearing fruits for the most part with scanty and inedible pulp and many seeds, the latter being ground and used as a substitute for flour, and of which the stem of the flower-bud is also eaten; but there does not appear to be an indigenous African species with fruit pulp capable of being utilised as a wholesome food. There is, however, no difficulty in accepting the idea of the spread of the plantain from Egypt to the greater part of the inhabited tropical Africa in very early times.

In whatever manner the introduction of the plantain among the savage nations of Africa originated, it is clear that it must have been known to them for many centuries, and that its cultivation by them has made it possible for some to take up their abode in the densest forests, far remote from the sea coasts and without dependence upon field crops, cattle or other sources of food supply apart from purely forest products.

The banana, the name properly applied to the saccharine form of the species above mentioned, is considered to have been a much more recent introduction into Africa. In this connection it should be mentioned that much confusion has arisen, owing to the misuse in India and Europe of the name plantain—bananas being frequently so-called in these localities. It is improbable, moreover, that plantains have ever been offered for sale in Western Europe; they are not generally used except as a vegetable, and all the sweet kinds of fruit called plantains in Europe and Asia are undoubtedly bananas.

At the present time the plantain is universally cultivated in the tropics, and in many places constitutes the staple food of the forest inhabitants of Africa; the banana being still uncommon or even unknown in some localities.

In the lake region of the Upper Nile, SPEKE says that the plantain constitutes the food of the countries one degree on either side of the equator; acres of ground being found covered with groves of it.

BURTON and SCHWEINFURTH have both referred to the dependence placed upon the plantain as a source of maintenance for the existence of tropical African tribes.

STANLEY, moreover, in *Darkest Africa*, refers to the large clearings he found in the great forest of the interior planted with plantains, and to the gigantic dimensions attained by some specimens of the fruit, which he records as up to 22 inches in length and 2½ inches in diameter.

Until recently the Ashantis, a fierce and warlike tribe of the Gold Coast hinterland, were unaccustomed to carry on any kind of farming work, and when without opportunity for, or success in, plundering their more industrious neighbours, subsisted for the most part on a diet of cooked plantains. With the exception of a few bulbous roots of *Colocasia antiquorum*, grown almost unattended on the swampy banks of the forest streams, and a few peppers springing up around their houses, the plantation of plantain suckers in the less dense parts of the perpetually damp forests, constituted the whole extent of their agricultural activity. The density of the forests themselves, as well as the protection and seclusion afforded by their presence, precluded any efforts to make large clearings. It is true that they obtained a few other foods of minor importance from the forest itself, but, from the time when they separated themselves from the Fantis, and adopted an

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independent forest life, their staple food must have been plantains, in the drying and storage of which they are still adept. From the streams in some parts they were able to obtain a few fish, and palm oil was easily extracted from the fruits of the wild *Elæis guineensis*, but in the dense forests, which formed their abode, no cattle were kept and their domestic animals consisted of a few fowls, while game, whether birds or beasts, was not at all plentiful.

The Ashantis were, apparently in early times, induced, by the presence of gold within the forest region, to penetrate deeper and deeper into the interior, and to dissociate themselves entirely from the coastal tribes, except when they raided them to procure slaves to work in the gold pits. They are said to have even altered their language, so that, although themselves understanding Fanti, they could not be understood by the Fantis. Their diet of plantains cooked in palm oil with the addition perhaps of a few wild herbs, seems to have constituted for them a complete food. In more recent times, owing to the suppression of their independent power, they have come into friendly and closer association with their neighbours and have adopted yams, cassava, maize and millet. Although not cultivating these themselves, they are able to obtain them in exchange for kola nuts and other valuable products of their forests. It is not uncommon, except in the north and centre of Ashanti, to find rice now freely used, although this is an import only of recent years from other countries

In preparing the plantain for food, the fruit is taken while it is still green, and is carefully peeled; the interior pulp is then split with a wooden blade and the pieces laid out in the sun to dry. The process is usually complete in about a day, when the product is generally of a chalky whiteness, and is very brittle. Ripe bananas, when treated in this same manner, never attain the same consistency, and always remain spongy, and become discoloured, due to the excess of saccharine matter contained in the pulp. Indeed, the dried bananas of the West Indies and East Africa are usually for this reason termed "banana figs." The dried halved plantains prepared by the Ashantis are convenient for storage, and can be kept for a long time. When required for food, they are broken or ground up and made into balls, constituting the "fufu" of the country. These balls are cooked in palm oil, extracted by boiling from the fresh fruits of the tree.

The use of the banana and plantain as a staple food is not confined to tropical Africa. JOHNSTON and CHURCH, writing of tropical America, refer to these fruits as among the principal foodstuffs of the native population, and say: "they are so extensively consumed as almost to take the place of cereal grains as a common article of diet." In an unripe state, cooked, they are said to supply the staple food of millions of people—"about six and half pounds of the fruit or two pounds of the dry meal, with a quarter of a pound of salt meat or fish, forms the daily allowance for a labourer."

FAWCETT, in his work on the banana, quotes from DAMPIER'S *New Voyage round the World* (1679-91) the different methods employed in the West Indies at that time for using the plantain as food, and gives DAMPIER'S statement that "on this fruit alone many thousands of Indian families in the West Indies had their whole subsistence."

Again, the same author, referring to Dr. WRIGHT'S "Account of the Medicinal Plants growing in Jamaica" (*London Medical Journal*, Vol. VIII., 1787, p. 272), quotes the latter as follows: "Plantains and bananas are eaten by all ranks of people in Jamaica, and, but for the plantains, the island would scarcely be habitable, as no species of provision could supply their place. Even flour or bread itself would be less agreeable and less able to support the laborious negro, so as to enable him to do his business or to keep his health."

Dr. HUTCHISON'S *Food and the Principles of Dietetics* gives an analysis of banana flour, compared with that of wheat, and shews that the former is richer in carbohydrates and mineral matter, but poor in proteids. He says that it would be found to be about equal to rice flour in nutritious value. It seems, however, to have greater advantages than the latter, judging by the more robust types of mankind produced upon a diet of it. Many authorities seem to agree that the starch found in bananas is peculiarly readily dissolved and digested in the alkaline juices of the body, the flour, for example, being easily soluble in the saliva.

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Remarkable evidence with respect to the digestive facility of bananas is shewn by the fact that it is not uncommon to observe small native children in India and tropical Africa, scarcely old enough to take any nourishment other than their mother's milk, given ripe bananas to suck to keep them quiet, and without apparently producing any bad results.

Some recent investigations on the nutritive value of the banana have been contributed by K. SUGUIRA and S. R. BENEDICT to the *Journal of Biological Chemistry* 36 (1918), No. 1, pp. 171-189 and 40 (1919), No. 2, pp. 449-468. After a series of experiments, the authors shewed that the banana was deficient in protein and in water soluble vitamin as a food stuff for the growth of white rats, and suggested as a conclusion from their experiments, that a combination of bananas and milk in proper proportions constituted a complete food.

W. BURNS and P. G. JOSHI, in the *Agricultural Journal of India*, Vol. XV., part ii., March, 1920, give an interesting account of experiments made in drying bananas in the Bombay Presidency. As a result they appear to have obtained a saffron, red or black coloured product, due presumably to their having used a ripened fruit belonging to a variety containing a high sugar content. The dried product they describe as "a most delicious sweetmeat eaten raw."

Under the title of "The world's widest known fruit," O. W. BARRETT, in the *Philippine Agricultural Review*, July 1912, p. 380, writes in support of the enhanced value of the cooked fruit as follows :—

"*Plantain or cooking banana.*—Throughout Africa and tropical America, some form of plantain is more often used as food (cooked) than probably any other variety of the family in those regions. Raw banana is not an ideal food, the content of starch and pectin is in such a form that the human stomach cannot digest it. Even unripe bananas and plantains become digestible when boiled or baked or even fried over a slow fire. It fact it might be roughly estimated that the ordinary ripe banana is rendered five times more valuable as human food, by cooking. A great deal of study is needed to determine the comparative nutritiousness of bananas and plantains."

In the *Journal of the American Medical Association*, Vol. LXXV., No. 6, pp. 417, 418, there is a short article with reference to the dietary value of fruits, and it is pointed out that the banana is, in comparison with citrus and some other common fruits, lacking in pronounced vitamin potency. This, however, does not affect the main issue ; the fact that the banana, or, I would prefer to say, the plantain, can be utilised as an almost complete food where citrus or the other common fruits cited could hardly occupy any such position, make their comparison, upon the food value basis, out of place.

In respect to the economic point of yield of food value for a given area of land, the plantain and banana stand high. A few estimates may be given in support of this, but their accuracy cannot be vouched for. HUMBOLDT stated that the banana could produce more food per acre, per year, than any other economic. It has been remarked that this statement may be justly questioned, but, that there is little doubt that the banana is one of three most productive food plants the world has ever known. O. W. BARRETT, previously referred to, states that a hectare ($2\frac{1}{2}$ acres), planted with bananas is estimated to yield 133 times as much food as the same area of wheat or 44 times as much as the area of potatoes. Unfortunately he omits to state how these figures were arrived at. Suffice to say that the productive power of a plantation of bananas or plantains under the most suitable conditions of soil and climate and with good cultivation is extraordinarily high, and as a plantation crop, the former fruit, in localities not infected with one of the destructive diseases the plant is subject to, is most remunerative.

It is scarcely possible to discuss the banana without making some reference to the enormous export trade in the fruit from the West Indies aud Canary Islands, which has sprung up since the beginning of the century, and necessitated the provision of a number of steamers specially in its service. This trade at one time enabled bananas to be placed on the English market at so low a price that they were rapidly becoming a common food among the poorer classes in Liverpool, and even in London. Sir JAMES CRICHTON BROWN, M.D., F.R.S., on his return from Jamaica, is reputed to have said in a speech

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delivered in England that "the banana, in the guise of a cheap luxury, is a substantial addition to our food supply, which is certain more and more to commend itself to the working classes in our large towns."

Dr. ROBERT HUTCHISON, in *Food and the Principles of Dietetics*, gives the analysis of flour made from dried unripe bananas as under:—

Moisture ...	13·0	Carbohydrates ...	80·0
Proteids ...	4·0	Mineral matter ...	2·5
Fat	0.5		

So far, the example has been given of a fruit which grows in tropical moist climates, where it is nearly always possible to produce grain crops, or where the inhabitants have been usually able, if they so desired it, to find other foods to substitute for it. As a contrast to this, the date may be selected as constituting the main and almost entire subsistence of a large number of nomadic peoples whose life is chiefly spent in travelling across the arid tracts of Africa and Asia, where there is little or no rainfall, and practically no vegetable or animal food obtainable. The date palm is not itself an inhabitant of these desert tracts, occurring in them only in the oases or sites of fresh-water springs, often separated by wide intervals of barren sand and rock.

It is, perhaps, true that without the existence of dates much of the history of the world would have been very different. The date has made possible the emigration of people from one part of the world to another, even where such formidable barriers to travel existed as those constituted by the expansive desert regions.

The dried date, which is the condition in which the fruit is of the highest utility, is a compact form of nourishment of the most digestible kind, and has enabled the Arab of Western Asia and North Africa, as well as the Persian, to subsist for long periods without any other food. Dr. CHARLES BENNETT, writing on the food value of the date, mentions that these people have found the date an ideal food, with just sufficient padding to favour intestinal function, carrying predigested material, ferments that aided its own digestion, and an attractive flavour. From personal experience of the dried fruit carried by the desert riders of the Western Sudan, I must admit that the flavour, to my taste, is about as attractive as that of a piece of leather, although the other qualities mentioned I am willing to concede. The author mentioned above remarks that an active, hard-working desert-dweller ate pounds of such dried dates in a day, and they formed almost his sole food for many months, with the occasional addition of a small amount of meat, milk or rice. It appears that to the man who consumes two pounds or so of dried dates as the bulk of his day's ration, little additional protein is necessary, and a sole date dietary is sufficient for the activity of many Arabs for long periods, without any additional food.

According to ATWATER (U.S. Dept. of Agric.), the composition of dried dates is as follows:—

Moisture ...	13·8	Carbohydrates ...	70·6
Protein ...	1·9	Mineral matter ...	1·2
Fat	2·5	Fibre	10·0

A pound of dried dates has a calorie value of 1275.

In the Western Sudan, the store of dried dates carried by the desert people seems to be frequently supplemented by a small amount of ground nuts, millet grains and the inevitable kola nut, though the dried date appears to form the basis of their diet.

Although the date is employed so largely as a sole food by certain desert inhabitants it does not always appear to suffice as a means of healthy human development. C. M. DOUGHTY, in *Travels in Arabia Deserta*, is insistent that the date-eaters of the Hejaz and the northern parts of Nejd, can be easily recognised from the itinerant Beduin, who habitually add goat or camel milk, and occasionally the flesh of these animals, to their otherwise date diet. The author mentioned, in referring to the Fukara tribe who are nomads with a range in the north of Nejd, says that among these date-eaters there was scarcely a well-grown man, beside the sheikh and his sons, or a comely woman. He further says the Arabians inhabit a land of dearth and hunger, and that there is no worse food than the date on which they are compelled to live in their few irrigated valleys. The

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fruit he says is overheating with too much "cloying sweetness" and does not supply enough "brawn and bone." All date eaters, he continues, are of a certain withered visage; especially the poorer inhabitants of Nejd. While well-faring people from the same oases have a pleasant and honest aspect, the people who subsist entirely on dates themselves say that it causes human nature to decay, and that this is aggravated by the addition of the luke-warm ground water, which is seldom wholesome, and is all they can obtain to quench their thirst. Nowhere in Arabia, says the author, does there appear such an "improbity of facial lineaments" as that seen in the township of El Ally (to the north of Medina), although in this place the best dates in the country are found. These inhabitants he says are deficient in bone, their upper face is sunken and flattened, and the jaw he describes as nearly brute-like and without beard.

Dates exist in cultivation in a very large number of forms, some of which are eaten fresh, others semi-dry, and others again dried to the extent mentioned above. The two former types are never used as food on journeys owing to their much greater bulk, and are only eaten by the inhabitants of the oases or tracts in which they are grown.

Speaking of the date-palm, P. B. POPENOE (*Date Growing in the Old and New World*) says it "is something more than a fruit tree which furnishes the principal means of existence to hundreds of thousands of people. To the Arab it is a sacred institution, identified with the Semitic race since the dawn of history, and consecrated by Mohamet both in his public and private life." The prophet is said to have subsisted for days together, during his years of poverty at Medina, on nothing but dates, washed down with water—a diet which, it is pointed out, is a common one still employed by thousands of nomads every year.

Phœnix dactylifera is the botanical name employed for all the varieties of date-palm which yield the edible fruits mentioned above, and, in common with other palms, the flowering spike is tapped for palm wine in certain parts of the Sahara, where in spite of the injunctions against the indulgence in intoxicants imposed by the Mohammedan religion, the juice is allowed to reach a highly fermented condition before it is drunk.

The preparation of dates for consumption as dessert, etc., gives employment to a very large number of people in Egypt, North Africa generally, Arabia, and Mesopotamia, and although in some cases the fruit is prepared in a careful and cleanly manner, in others, notably in the case of the mashed dates termed "Agwa" in Egypt, the method of preparation is uncleanly and nauseating. "Agwa" is frequently seen for sale in the streets of London, but the proportion of insect life sacrificed as a make-weight in the bulk is, perhaps fortunately, unknown to the consumer.

Few other fruits can be said to occupy the place of a main food, though several enter largely into the diet of some of the world's inhabitants. The bread fruit (*Artocarpus*), the papaya (*Carica papaya*), the coco-nut (*Cocos nucifera*), are examples of some of these, while some other fruits, less well known, have been shewn to contain matter of high nutritive value. The avocado pear (*Persea gratissima*), for instance, seems to be prominent in this respect, although from the nature of its growth it is hardly likely to become an important food. The analysis of the pulp of this last-named fruit is, according to WOOD and MEWILL, as follows:—

Water ...	81·1 per cent.	Carbohydrate ...	6.8 per cent.
Proteid ...	1·0 ,,	Mineral matter ...	0·9 ,,
Fat ...	10·2 ,,		

These few examples may serve to indicate in some measure, how a particular fruit has, by contributing so largely to the nourishment of the human body, influenced human development in the world's history.

DISCUSSION.

Dr. D. E. ANDERSON: I am glad to have heard Mr. DUDGEON'S paper, as I have travelled in many lands where bananas and plantains grow luxuriantly. There are different species and varieties: the commonest in Mauritius are the gingely, naine, mignonne, sinneroe, malgache, and ollier. The last two are best eaten cooked, fried or baked in hot

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ashes. The gingely is very small, and is *recherché*, but when eaten too freely at night is said to produce cholerine; in fact, most plantains, if not quite ripe are apt to be indigestible when eaten at night, especially if partaken of with milk.

As we are all aware, the late Sir Alfred Jones, of Liverpool, was greatly instrumental in reducing the price of bananas in this country by starting a fleet of steamers between the West Indies and Liverpool, to bring bananas, coco-nuts and cocoa to the British Isles. He thus encouraged a variety of industries, and opened valuable markets between our Western Colonies and the mother country. Before the war a large number of vessels could be seen in the harbours of Jamaica, Trinidad and British Guiana, loading fruit for North America and this continent. Seeing that the West Indies are only seven or ten days from our shores, and that the banana is such a nutritious and acceptable fruit, that margarine and other oily products of daily use can be obtained from the coco-nut, that the soil and climate of those islands are suitable for the growth of the cocoa and the pineapple—it struck me, whilst I was visiting that part of the world seven years ago, that we were not taking the fullest advantage of the riches nature had placed at our disposal. What is needed is not only more capital to develop the plantations of the above-named fruit-trees, but greater and more rapid means of transport.

There are many other fruits of minor importance which would be welcomed in this country could they reach us in six or seven days after having been plucked ripe off the trees, such as the mango, the custard apple, the avocado or alligator pear, the letchi, the guava, the "fruit de Cithère," the sour sop, the loquat, the jack and the papaw, and different varieties of nuts, among others the cashew and the badannir, which in the tropics replace the chestnut and the filbert nut at dessert. There are delicious conserves and comfits to be obtained from some of the cooked fruits, and "pâtes" and nougats made with guava, the mango and the papaw; but the demand in Europe for these luxuries is not great enough to encourage the native confectioners to export them. I must not sit down without referring to the refreshing drinks or lemonade made with the ripe tamarind pods, or from the water inside the tender nut of the young coco-nut.

Dr. A. Balfour.: As Mr. Dudgeon has indicated, important work on the banana has recently been conducted in the U.S.A. I do not think, however, that he mentioned that by Pease and Rose, who shewed that the digestibility of the raw banana was directly proportional to the ripeness of the fruit. No deleterious effects follow the ingestion of large amounts of fully ripe bananas, but when eaten unripe unpleasant symptoms may develop. The banana should not be eaten raw until after brown spots appear on its skin. From the sanitary standpoint interest attaches to the peel of the banana, which protects it from contamination. When at Mwanza, at the southern end of Victoria Nyanza, during the war, I was told by a Greek that the Germans had established quite a large trade in the preparation of what was called "banana figs." These were made by splitting and drying the ripe fruit, which was then coated with sugar and exported as a delicacy. Nothing of this kind had been accomplished in Uganda, and I think it a matter which our British Colonies might well consider. It is interesting to note that, during the war, banana meal largely took the place of wheat flour in the prepartion of bread. It is said to have produced a saving of some 30 per cent. of wheat flour, and proved quite palatable.

Mr. Dudgeon mentioned the avocado or alligator pear, and indicated the analysis of its pulp. It is interesting to note that recent work by Condit and Jaffa has shewn that it may possess as much as 20 per cent. of fats, and it has been suggested that it may prove valuable as a food for diabetics. Moreover, experiments have shewn how excellent is the utilisation of avocado fat in the body, even when as much as 4½ oz. a day are consumed. Ordinary cream rarely exceeds the avocado in available fat content, and the fruit is likely to fill a felt want in cases where a palatable fat is required.

Unfortunately, we rarely see it in this country, as it does not stand transport well, but it is probable that it will come much more largely into use in the United States than is at present the case.

Dr. D. G. R. Black: I would like to make a remark with regard to one fruit which is mentioned at the end of this valuable paper, and I am sorry Mr. Dudgeon did

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not give more particulars with regard to it, as it is one of the best and one of the most valuable of all tropical fruits. Those of us who have lived in the countries where it is abundant realise its extreme value as an article of diet. The fruits he has dealt with, particularly the banana, are, of course, of vast importance, but mainly in the smaller and more sparsely-populated parts of the tropics. What interests us, as Fellows of this Society, more than that, is the question of fruits in relation to dietary in the densely-populated tropical and sub-tropical countries. The fruit to which I allude is the pawpaw, or *Carica papaya*. We know the active principle is papain, and we know it has high digestive value, and is of immense value in our most densely-populated tropical possessions. There are many parts of the tropics in which a large proportion of the diet of the inhabitants is either derived from the sorghum or millet series or the legumes, or even maize. We know that the legumes are not readily converted, and if you take sorghum conversion, it does not amount to more than half of the proteins We have seen the very unsatisfactory development of the young growing native child. That, in most cases, is due to protein shortage in its diet. The protein content of these foods it not, in itself, sufficient ; and if the human being can only utilise about half the protein content, the consequence in development is obvious. Secondly, a very large proportion of the intestinal troubles which exist in those countries are—and I think Dr. CASTELLANI is of the same opinion—attributable largely to the unconverted proteins from these sources in the intestinal canal. What I suggest is that, not only out there, but even in this country, an extensive use of the pawpaw, with its marvellous digestive ferment, would get rid of the defective power of converting those proteins, and so remove the two factors—the inadequate nutrition of the growing child and the intestinal disturbances of the adult. I suggest to people who indulge in more proteins than can be converted by their pepsin and their pancreatic secretion, and who thus send down their alimentary canal a large stream of unconverted proteins, that they should eat pawpaw with their meals. In many cases which have come under my notice, pawpaw taken with those foods in the tropics averts troubles in both natives and Europeans. And where I have to deal with chronic cases of beriberi whose digestion was greatly impaired, they recovered in half the time if the diet comprised a sufficient quantity of papaya food.

The PRESIDENT: On behalf of the Society, I propose a vote of thanks to Mr. DUDGEON for this most interesting and instructive paper. He has shewn us the value of the plantain, the date, and other fruits. There is one matter I hardly understand, and that is what was mentioned by Dr. ANDERSON, namely, that a mixture of banana and milk may produce the bad effects which were spoken of. It might possibly have been that it was due, as Dr. BALFOUR pointed out, to the possibility of the bananas taken with milk being unripe. In India the Hindoo widows like to be stout, and they accomplish it by consuming bananas and milk. It may be that the banana spoken of was of a different species. I feel inclined to think it was the milk which was doing the harm. I do not know about the West Indies, but in all countries I have visited I have found that the milk supplied is not pure; when it is obtained from the milk-seller, his product is as bad as that of the milk-seller in the West, and the adulterant he uses is not always pure water. One has not had the opportunity of being with tribes who live entirely on a fruitarian diet, but I remember once meeting, in Ceylon, a German cavalry officer who was a fruitarian. He had found it was too expensive to live in Berlin on fruit; it cost him about 30s. a day. Therefore he went to the Fiji Islands, and there lived on fruit for a considerable time. He got dissatisfied with the conditions there, and came to Colombo. There he had bought a coco-nut farm, and he lived on coco-nuts and other forms of fruit, and a finer looking man one could not see anywhere There is no doubt that in Europe we are too much inclined to think that the only growth-producing food is meat, which, it is thought, ought to be taken in large quantity. As a matter of fact, it is only within the last half-century or so that this meat-eating fashion has come into vogue. If before this time we take Europe as a whole and consider the diet of the majority of the people, we find they were mainly vegetable-eaters, with meat only occasionally entering into their diet, and sturdy folk they were.

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TRANSACTIONS

OF THE

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

DECEMBER 17TH, 1920, AND JANUARY 21ST, 1921.

VOLUME XIV. No. 5.

Proceedings of a Laboratory Meeting of the Society, held on Friday, December 17th, 1920, at The London School of Tropical Medicine, Endsleigh Gardens.

LABORATORY MEETING.

Dr. A. BALFOUR exhibited a number of models of Indian fruits and food-stuffs prepared in the Bazaar at Lucknow. He drew special attention to the model of the Indian chapatti, and remarked that a large number of these unleavened cakes found their way into the incinerators during the campaign in Mesopotamia, the Indian ration of atta, from which they were prepared, being in excess. On the other hand, there was at first a shortage of anti-scorbutic elements in the diet, and in this connection he mentioned the model of tamarind. Tamarind and cocum supplied a certain amount of anti-scorbutic vitamines to the diet of the Indian Troops, but they possessed only slight value.

The models shown were furnished with descriptive labels and represented a large variety of the more common Indian fruits and bazaar food-stuffs.

Dr. P. MANSON-BAHR exhibited a series of microscopic preparations and drawings illustrating the appearances, through the sigmoidoscope, of dysenteric lesions of the lower bowel.

The value of this instrument as an aid to diagnosis in these conditions was emphasised, for by this means one is able to distinguish between the scarring and granulation tissue resulting from bacillary dysentery and the definite ulceration and hæmorrhages of the amœbic disease

In scrapings of the ulcerations, obtained by means of a match affixed to a holder and inserted through the sigmoidoscope, it has been possible to demonstrate active *Entamœba histolytica* in large numbers; in one particular preparation thus obtained and exhibited fifty-six active entamœbæ were visible under one field of a one-sixth inch objective, while neither these organisms nor their cysts had been discovered in the stools

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of the patient. The bearing of this work upon subsequent treatment of the case was emphasised.

Other preparations consisted of a series of sections of subtertian malaria tissues from Palestine, showing the enormous number of parasites in the blood-vessels, especially those of the heart.

A section of a stomach of *Anopheles maculipennis*, containing large numbers of oocysts of benign tertian malaria—a specimen obtained in the London School of Tropical Medicine during the past summer—was also shown.

There were a few microscopical preparations of cirrhotic changes in the liver of kala azar, and also demonstrating the persistence of the Leishman-Donovan bodies in the bone-marrow from a recent case in the Tropical Diseases Hospital.

Two clinical cases of liver abscess illustrated different aspects of this disease :—

The first man had an abscess of the right lobe, which was aspirated through the epigastrium and 1½ pints of pus removed by Col. J. J. PRATT. No open operation was performed, and a perfect recovery has resulted.

In the second case, an abscess of very considerable standing—possibly as long as seven years—had burst into the right bronchus; great improvement and apparent recovery had taken place on large doses of ipecacuanha. On admission to hospital he had exhibited the stigmata of grave pulmonary involvement—cyanosis, clubbed fingers and toes, together with profuse blood-stained but not offensive sputum. Owing to long-continued septic absorption the patient developed thrombosis of both internal saphena and iliac veins; in consequence of which a compensatory venous anastomosis on the abdomen and thorax had arisen.

Dr. G. C. Low showed a series of specimens of filarial embryos illustrating the different varieties commonly seen in human blood. An interesting specimen of a living and active *Filaria bancrofti* embryo, from a case under treatment with tartar emetic injections, was also shown. Though the patient had had 13½ grains of the drug in all, yet the number of embryos still continued the same, and, as the living specimen showed, had suffered no diminution of vitality. Dr. Low stated that another case under similar treatment showed no beneficial effects of the drug either, and he was doubtful of its being of any use in such infections.

Dr. E. E. ATKIN and Mr. A. BACOT gave a demonstration of the organisms known as Rickettsia bodies, thought to be the cause of typhus fever, trench fever and Rocky Mountain spotted fever; also sections from the brains of guinea-pigs infected with typhus fever showing lesions in small blood vessels.

The following preparations were exhibited :—*Rickettsia prowazeki* in human lice (*Pediculus capitis*) and monkey lice (*Pedicinus longiceps*), *R. quintana* in human lice (*P. corporis*), *Dermacentroxenus rickettsi* in the tick (*Dermacentor venustus*), *R. melophagi* in the "sheep ked" (*Melophagus ovinus*), and a new species of Rickettsia from the Malpighan tube of a bed bug (*Cimex lectularius*).

Lieut.-Col. G. E. F. STAMMERS exhibited specimens illustrating spirochætal jaundice in the guinea-pig. The virus was obtained from wild rats in London and elsewhere in England.

The exhibit consisted of the following :—

(1) Guinea-pig No. 35 (male) inoculated 8th December, 1920, intraperitoneally with mixed kidney, liver and lung emulsion of another guinea-pig; found dead 16th December, 1920. Post-mortem appearance of intense jaundice of skin and internal organs; hæmorrhages in lungs, skin, axillary, inguinal, pubic and lumbar regions; also in the testicular area.

(2) Guinea-pig No. 36 (female) inoculated as above on 10th December, 1920; killed 17th December, 1920. Post-mortem appearance similar to the above.

(3) Chart of inoculations commencing with rat No. 147, obtained from

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Hammersmith, on 15th November, 1920, through a series of guinea-pigs to those exhibited, which were of the fourth passage. The temperature records of the series of guinea-pigs were attached.

(4) Microscopic specimens of :—

(*a*) Leptospira in the kidney of rat No. 147.

(*b*) Leptospira in the lung of guinea-pig No. 21, which was in the series of inoculations above mentioned.

(5) A mounted specimen of guinea-pig's lungs, showing hæmorrhages.

(6) A colour transparency of a guinea-pig dead of the disease, showing especially hæmorrhagic areas in the mesentery. This guinea-pig was from another series of transmissions from rat No. 111, carried out in November.

Dr. HUGH S. STANNUS showed (1) photographs of a series of cases of splenomegaly with enlargement of the liver of unknown etiology from Central Africa. Isolated cases had been seen in various parts of Central Africa, but the cases under consideration were drawn from the native population of a group of villages near the north end of Lake Nyasa, among whom the affection was found in something over 50 per cent. of the children.

Dr. NORMAN LEYS, who had observed one or two cases over a long period, was asked to investigate this group, and his observations were published in the TRANSACTIONS of this Society, Vol. X., page 67. His findings were negative, and covered a very limited number of cases. Dr. STANNUS could offer nothing in the way of commentary, but stated that the photographs gave a very good idea of the variety in size, absolute and relative, of the spleen and liver in this clinical entity.

(2) Photographs and microscopical preparations of cases of malignant disease seen among natives of Central Africa. Little is with certainty known about the incidence of malignant disease among native races. That they are rare cannot be doubted ; in fifteen years, with large opportunities for observations, he could not record a single case of carcinoma of the breast in a woman or epithelioma of the lip in a male. On the other hand, carcinoma and sarcoma are seen, and it would be of value if all such cases were recorded with a view of throwing any light on the etiology of those diseases.

The cases shown were (*a*) spheroidal carcinoma of the thyroid gland in a woman aged 50 ; (*b*) primary carcinoma of the liver in a youth aged 16 ; (*c*) myxo-endothelioma of the parotid gland in a young male adult ; (*d*) myxo-chondro-endothelioma of parotid gland undergoing carcinomatous change ; (*e*) round-celled sarcoma of testicle, with secondary growths in both orbits.

Surgeon Rear-Admiral P. W. BASSETT-SMITH showed specimens illustrating the infection of the guinea-pig fœtus with trypanosomes. They had been found in—

(1) The liver of a fœtus from a parent infected with *T. rhodesiense.*

(2) The liver and spleen of a fœtus from a parent also infected with *T. rhodesiense.*

(3) The placenta of two parents infected with *T. rhodesiense.*

(4) The blood of two young guinea-pigs born on 15th September, 1919, of a parent infected with *T. gambiense.* They grew healthily till 4th February, 1920 (five months), and 14th March, 1920 (six months), when they died. Trypanosomes were first seen one month after birth. The parent died with a ruptured spleen four months after inoculation. The young guinea-pigs thus lived longer than the mother after inoculation. The placental blood in which trypanosomes were found shewed many free nuclei, flagella, and centrosomes, as well as entire trypanosomes.

A table, illustrating the increase in virulence of *T. gambiense* during ten passages through the guinea-pig, was also shown. The first guinea-pig survived 23 months after inoculation from the human case, while in the succeeding passages the animals died in 5, 7, 6, 5½, 5, 4, 3, 1¾ months respectively.

Guinea-pigs born of an infected and a healthy parent were transposed with a view of testing the infective possibilities of suckling. Two guinea-pigs born of an infected parent were both infected, but the healthy mother which suckled one of these did not acquire

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an infection. The guinea-pig born of the healthy parent did not become infected, though suckled by the infected parent.

Mr. ALDIS gave a demonstration of the new Aldis-Microprojector, an apparatus designed for projecting on to a screen microscopic objects. The first exhibit consisted in the projection of the whole field of a live cell containing water-fleas, cyclops, and other larvæ, which could be clearly seen swimming about in their native element. This was kept on the screen for fully ten minutes, without any of the animals showing great distress from heat exposure.

A single water-flea in a sunk cell was then substituted at the higher magnification of 500. The beating of the heart, contractions of the eye-muscles, and other details of internal organs were clearly visible. Finally, a blood preparation at a magnification of 1,750 diameters was projected on the screen, illustrating the value of the apparatus for teaching purposes. In every case Mr. ALDIS was able to demonstrate the extremely small amount of heat communicated to the microscope slide—a condition rendered possible by the small candle-power of the lamp used, which, however, did not detract from the good illumination.

It was explained that the actual apparatus used in the demonstration was only experimental, though all the essential features of the finished product which he would put upon the market were there, except for an improvement which was being introduced with a view to simplifying the adjustment of the electric lamp. The latter was run off the ordinary electric lighting circuit, with an adjustable resistance in series. The whole apparatus could be easily carried about in a hand-bag, and was ready for use as soon as the necessary connections had been made to the electric lighting circuit. (Any enquiries should be addressed to ALDIS BROS., Sparkhill, Birmingham.)

Dr. A. C. STEVENSON showed specimens illustrating the extravascular distribution of trypanosomes.

(1) A section of a mediastinal lymphatic gland of a rat infected with *Trypanosoma rhodesiense*. The lymphocytes are replaced by a mass of trypanosomes, whereas few are seen in lymph channels and blood-vessels.

(2) Section of a fœtal heart. The mother of the fœtus was killed when showing a heavy infection with *T. rhodesiense*. The sections show trypanosomes in masses lying between the muscular fibres of the papillary muscles

(3) A section through the internal ear of the same fœtal rat, showing a mass of trypanosomes in the areolar tissue surrounding membranous labyrinth.

(4) A section of kidney of guinea-pig infected with *T. nigeriense*. Trypanosomes are seen in the areolar tissue between the fat of the pelvis of the kidney and the main part of the kidney.

(5) A section of the stomach of a guinea-pig infected with *T. nigeriense*. The trypanosomes lie round the vessels near the surface of the mucous membrane. Trypanosomes can also be seen wandering between the epithelial cells. This massing of the trypanosomes round the vessels may be partly the cause of the ulceration of the stomach often observed.

(6) A section of choroid plexus of brain of guinea pig infected with *T. nigeriense*. The trypanosomes are very numerous in the areolar tissue and very few are seen in the vessels. A later infection would also show an extensive small round-celled infiltration of the same areolar tissue, and a practical absence of trypanosomes.

Major H. BROWN, C.I.E., I.M.S., gave a demonstration on the use of citrated media.

It was shown that, when the paratyphoid bacilli were grown on nutrient broth to which 1 per cent. of tri-sodium citrate had been added, the growth of *B. paratyphosus* A was inhibited, whilst that of *B. paratyphosus* B and C was markedly enhanced.

Also *B. paratyphosus* C formed gas in this medium.

Similarly the true cholera vibrio shows an enhanced growth in this medium, whilst that of the spirilla of METCHNIKOFF and also of FINKLER PRIOR is inhibited. This simple

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medium should prove useful in the differentiation of the paratyphoid group of organisms, and also in the case of certain cholera-like vibrios.

Standardisation of bacterial suspensions by opacity.—A series of opacity tubes, containing known quantities of barium sulphate suspended in 1 per cent. solution of citrate of soda, were shown. These tubes corresponded in opacity to that of definite dry weights of bacterial substance per cubic centimetre. It is claimed that by this method a vaccine can be more rapidly and more accurately standardised than by the majority of methods in common use, and that the correlation of opacity to weight of dried bacterial substance is a far more accurate method of expressing dosage than when it is denoted in terms of the number of organisms contained in vaccine.

Dr. J. G. THOMSON gave a demonstration illustrating leishmania infections. The following specimens were shown :—

(1) A living culture of *Leishmania donovani* isolated in N.N.N. medium from spleen puncture material.

(2) A living culture of *L. tropica* isolated in N.N.N. medium by Dr. C. M. WENYON from a case of oriental sore.

(3) A section through a non-ulcerating oriental sore, showing *L. tropica* within the macrophages.

(4) A section of the spleen of a case of kala-azar, showing *L. donovani* within the macrophages.

(5) A smear from an oriental sore, showing *L. tropica* within a macrophage.

(6) A smear from spleen puncture material from a case of kala-azar, showing an extremely heavy infection with *L. donovani*.

(7) Stained preparations of the cultural forms of *L. donovani*, showing flagellates of the leptomonas type.

The sections and smears were stained with Giemsa's stain, and showed clearly the morphology and distribution of parasites in the affected tissues. It is of interest to note that the cultures of *L. donovani* were obtained from the bone marrow of a fatal case fourteen hours after death. Furthermore, the case had been treated with a considerable amount of antimony compounds.

Lieut.-Col. CLAYTON LANE, I.M.S. (retd.), demonstrated his levitation technique for the concentration of ankylostome eggs, which is briefly as follows :—

Resolve 0·5 c.c. of stool into its constituent parts by thorough shaking in water. Strain through wire gauze of 100 strands to the linear inch into a centrifuge tube consisting of a piece of plain glass tubing corked below. Centrifuge sufficiently to precipitate all ova. Pour off the supernatant fluid. Withdraw the cork. Transfer the attached deposit to a slide prepared thus. Draw a grease line across an ordinary slide half an inch from each end. Across an oblong dish 4in. wide, an inch deep and two-thirds full of water, place, parallel and nearly two inches apart, two straight, fine, flexible, polished steel ribbons, with ends turned down. Across the middle of these "sliders" place the slide bearing the deposit from the centrifuge. Add water with a pipette to form a deep meniscus, which will be bounded by the grease lines and the edges of the slide. Break up the deposit thoroughly by stirring. Stand for five minutes. With a depressor depress vertically the slide, supported as it is on the sliders. Draw the slide sideways along the bending sliders. On its emergence, considerably tilted, the surface tension removes practically all fæcal matter. Examine under the microscope with a considerable meniscus of water. With a loss averaging 16 per cent. ten times as many uncamouflaged ova are concentrated on the same area, as is the case when the ordinary centrifuge technique is used. It was emphasised that unless methods of concentration are checked, as levitation is, by sufficiently numerous comparative counts of ova, their reputed value rests mainly on unscientific, if enthusiastic, guess-work.

Professor R. T. LEIPER showed a series of microscopical preparations illustrating the distinguishing characters of various genera of flukes including Heterophyes, Metagonimus

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and Clonorchis. There were also (*a*) Eggs of *Ancylostoma duodenale* from the fæces of an experimentally infected cat. (*b*) Infections of Cyclops and Cypris with Cysticercoids. A number of rare helminthological works were also exhibited, including BURMEISTER's manuscript notes as a student under NITZSCH.

Dr. A. CASTELLANI showed specimens collected from cases of various tropical mycoses—bronchomycosis, Tinea imbricata, acladiosis and hyphomycetic cystitis. He called attention to the fact that cases of bronchomycosis are often mistaken for tuberculosis. He stated that the aspergilli, so often found in scales of Tinea imbricata, were not the true agents of the disease, the causative fungi being totally different organisms belonging to the genus Endodermophyton. Acladiosis is an ulcerative dermatomycosis, which is often mistaken for syphilis. It is due to *Acladium castellanii* (PINOY). Cystitis and urethritis of hyphomycetic origin are not very rare in the tropics.

Dr. B. BLACKLOCK gave a demonstration illustrating a case of indigenous infection with *P. falciparum*. The infection occurred in a girl of eighteen who had never been out of the British Isles; the patient died after a brief illness, the parasite being discovered after death. The following preparations were shown:—

(1) Smear of bone marrow containing gametocytes in various stages of development.

(2) Smear of spleen containing numerous segmenting parasites.

Blood film showing heavy infection of erythrocytes with young trophozoites, and a few gametocytes and segmenting forms.

Colonel S. P. JAMES showed a map illustrating the distribution of 426 cases of malaria contracted in England since August, 1917, and the following microscope slides:—

(1) A series of blood films showing young forms, sporulating forms, and gametocytes of *P. vivax* present in the blood of a child, aged seven years, who had contracted the infection at Colchester, Essex, in 1919. The notes and temperature chart of the case are given on page 112 of Colonel JAMES's book, *Malaria at Home and Abroad.*

(2) The stomach of a specimen of *A. maculipennis* which had been fed on a case of benign tertian malaria contracted, in 1917, at Sheerness, in Kent. Many well-stained zygotes, in different stages of development, were present in the preparation.

Mr. J. T. EDWARDS, Royal Veterinary College, London, assisted by Mr. S. C. J. BENNETT, showed the following pathological specimens of veterinary interest:—

(1) *Foot and Mouth Disease*—Lesion of ox's tongue, showing the typical shallow ulcers which remain after the blood-vessels have burst.

(2) *Swine Fever.*—A portion of the ileum of a pig, showing the mushroom or button-like raised necrotic areas preceding ulceration.

(3) *Swine Erysipelas.*—(*a*) Skin of a pig with very fine examples of the red diamond-shaped urticarious lesions found in the acute infection. (*b*) A pig's heart with valvular endocarditis as found in the chronic form.

(4) *Calf Diphtheria.*—Two specimens showing the lesions produced by *B. necrophorus*: the first a calf's muzzle with erosion of wing of one nostril and the nasal septum. and the second a longitudinal section of a calf's tongue with extreme necrosis of the dorsum.

(5) *Liver Abscess.*—A calf's liver with multiple "abscesses," or rather necrotic foci due to *B. necrophorus.*

(6) *Coccidiosis.*—Liver of rabbit with multiple small white spots—dilatations of bile-duct packed with coccidia.

(7) *Verminous Aortitis.*—Aorta of a horse with aortitis, showing worms (*Strongylus vulgaris*) in the inner coat and some adult worms on the surface.

(8) *Hodgkin's Disease.*—Enlarged spleen of a dog.

Colonel A. ALCOCK showed "Some Arthropods of Medical Interest."

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Proceedings of a Meeting of the Society held on Friday, January 21st, 1921, at 8.45 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Professor W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P. (*President*), in the Chair.

THE INFLUENCE OF HUMIDITY ON THE LIFE HISTORY OF MOSQUITOES AND ON THEIR POWER TO TRANSMIT INFECTION.

BY C. A. GILL, LIEUT.-COLONEL I.M.S.

The association of malaria with the humid atmosphere prevailing around swamps and marshes is one of the oldest epidemiological observations on record. It was remarked by Hippocrates, and the observation has been confirmed by almost all classical writers down to the present day. Modern treatises on malaria refer to the importance of humidity, but little or no indication is given in regard to its precise significance.

My excuse for appearing before you this evening is the fact that some recent observations and experiments appear, in some small measure, to fill this gap in our knowledge, and at the same time to suggest that meteorological conditions generally exercise an important rôle in malaria.

An account of the preliminary investigation will shortly be published in the *Indian Journal of Medical Research*, and I only propose to consider here, as briefly as possible, the rôle of humidity—and, to some extent, the influence of temperature—on the life-history of mosquitoes and on their power to transmit infection.

(1) THE RÔLE OF HUMIDITY.

It should be stated that humidity will be considered in this paper entirely from the point of view of relative humidity, which is, of course, the amount of moisture present in the air expressed as a percentage of the amount necessary to cause saturation.

It is also convenient to emphasise here the fact that relative humidity, owing to the physical properties of air and aqueous vapour, is subject to marked variations with change of temperature, so that a rise in temperature will, *cæteris paribus*, cause a fall in relative humidity, whilst a fall in temperature will be associated with a rise in relative humidity.

It thus comes about that an hourly chart of atmospheric relative humidity is normally somewhat U-shape, the humidity being low during the heat of the day and relatively high at night. But not only is relative humidity largely dependent upon temperature, but, owing to the fact that high relative humidity interferes with the radiation of heat, the atmospheric temperature is, in some measure, determined by the relative humidity.

The point arising out of this close interdependence of temperature and humidity is the fact that it is necessary to take into account both factors in any attempt to determine the influence of either of them in malaria.

As anopheline mosquitoes and human cases of malaria were not available the experiments to be now detailed were conducted with *Culex fatigans* and bird malaria (*Proteosoma grassii*).

The first point it was sought to determine has reference to the effect of varying degrees of humidity on the length of life of *Culex fatigans* and on its power to transmit bird malaria.

The procedure consisted in placing mosquitoes in a series of incubators at a constant temperature (27° C.) and determining their duration of life under different degrees of

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relative humidity. In experiments with proteosoma mosquitoes, which had fed on the previous night on an infected sparrow, were divided into batches of equal numbers and placed inside mosquito "hotels," which were then allotted to their respective incubators, so that, except in the case of humidity, all the conditions were identical.

During the progress of the experiments the "hotels" were examined each morning and dead mosquitoes removed and counted. In the case of experiments with infected insects, an equal number was removed for dissection according to a pre-arranged programme. In all infected specimens a careful count was made of the number of oocysts in the midgut, but it was not possible, except very roughly, to estimate the degree of infection of the salivary glands with sporozoits.

The relative humidity was calculated in the usual way by means of "wet and dry" bulb thermometers, one being placed in each incubator together with a maximum and minimum thermometer. Readings were recorded three times daily, and, from the mean thus calculated, the mean temperature and humidity maintained during the course of each experiment was determined.

To maintain high degrees of relative humidity (over 80 per cent.) a series of small wet sponges was placed in the incubator until the wet bulb temperature showed the reading corresponding to the required degree of humidity.

When low degrees of relative humidity were required the air in the incubator was first dried by means of calcium chloride, and then various dilutions of strong sulphuric acid were placed in the incubator until the wet bulb reading remained steady at the temperature corresponding to the required degree of relative humidity.

Finally, to obtain an approximate relative humidity of 100 per cent., air was first drawn through a wash-bottle containing water at the required temperature, and then through the mosquito "hotel" by means of a pressure-pump attached to the laboratory water-supply. Experience proved that, with the exercise of care and by keeping an eye on the wet bulb temperature, the above procedure enabled different degrees of relative humidity and a constant temperature to be maintained in the incubators without difficulty.

These experiments, of which a large number was performed, gave definite and consistent results, which will now be summarised.

It was found that with a relative humidity of 45 per cent. or less (at a temperature of 27° C.) *Culex fatigans* are invariably killed within five days, and that the lowest degree of relative humidity associated with the survival of these insects beyond five days was 48 per cent. On the other hand, with a relative humidity of between 65 and 75 per cent. death rarely takes place within ten days, whilst a relative humidity of over 80 per cent. is even more favourable to life. The effect of relative humidity for longer periods than ten days was not determined.

It was also found that under the above conditions of temperature and humidity—and irrespective of the degree of relative humidity—the development of *Proteosoma grassii* in *Culex fatigans* exhibited constant characters; *i.e.*, young oocysts were seen on the third day, mature oocysts on the fifth day, and sporozoits were first seen in the salivary glands on the sixth day, although heavy infections were not noted until the ninth day.

The influence of low degrees of relative humidity (45 per cent. or less) on the number of oocysts developing in the midgut and on their rate of development could not be ascertained, as mosquitoes incubated under these conditions were desiccated and could not be dissected.

With higher degrees of relative humidity (over 48 per cent,) no appreciable difference, either in the degree of infection—as measured by "oocyst counts"—or in the rate of development of oocysts could be detected as the result of varying degrees of relative humidity. On the other hand, it was found that a close relationship appeared to exist between the number of oocysts in the midgut of the mosquito and the number of parasites in the blood of the sparrow, provided the "parasite count" was made on the day of feeding.

It is therefore concluded:—

(1) A mean relative humidity of not less than 48 per cent. (at a temperature of 27° C.) is essential to the existence of *Culex fatigans* for a period of five days, and

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that any lower degree of relative humidity will cause the transmission of bird malaria to cease owing to the premature death of the insect-host.

(2) Relative humidity does not appear to exert any direct influence on the *degree of infection* or the *rate of development* of the sexual parasite of bird malaria in the mosquito.

In view of the influence of humidity on the length of life of *Culex fatigans*, it might be anticipated that it would also exercise an influence on their habits, in which connection the following observations were made.

It was noted in April and May, when *Culex fatigans* prevailed in Lahore in vast numbers, that large numbers of dead and dying insects could be found in exposed situations, such as open verandahs, whilst little or no mortality occurred amongst those inside houses, where in my own house they collected during the heat of the day chiefly in bath-rooms, in chimneys and amongst plants in the drawing room. By estimating the relative humidity, by means of "wet and dry" bulb thermometers, prevailing in these situations, it was found that, whilst in the verandah it varied between 30 and 40 per cent., it was at the same time in different parts of the house between 50 and 65 per cent.

In spite of wire gauze over all doors and windows, mosquitoes in large numbers gained access to the house, mainly in the early morning, and it was the custom to clear the house each evening by opening the doors of the main entrance, after switching on the light in the verandah and darkening the house.

The insects from all parts of the house would then stream out whilst none would attempt to enter. On the night of May 9th, however, which was an exceptionally hot dry windless night, the usual procedure was reversed—no mosquitoes attempted to go out whilst numbers began to enter the house.

During the course of the experiments unaccountable difficulties not infrequently occurred in feeding *Culex fatigans* on sparrows; sometimes for several nights in succession no insects would feed, whilst occasionally heavy feeds would be obtained. It was subsequently found from the laboratory records that when the daily mean humidity was less than 40 per cent., feeding experiments failed, whilst successful feeds were associated with a relative humidity of over 50 per cent. This observation, together with the well-known fact that mosquitoes, which apparently disappear in the Punjab during the intense dry hot period in June, suddenly reappear and commence to feed after the first downpour of monsoon rainfall, suggests that relative humidity exercises an influence on the feeding habits of mosquitoes.

Finally, it may be mentioned that although, so far as is known, the maximum length of life of *Culex fatigans* has not been determined, it is known that hibernating insects, under conditions which essentially comprise a low mean temperature and high mean relative humidity, may live for several months. These observations therefore suggest that relative humidity exercises a marked influence on the habits and life-history of *Culex fatigans*. It is not contended that anophelines are equally affected, nor that all species of mosquitoes will be found to exhibit the same degree of susceptibility to humidity, but it is a justifiable assumption that the conclusions reached in the case of *Culex fatigans* will be found to apply in principle to all mosquitoes.

In these circumstances the precise significance attaching to the predilection of "domestic" species for certain types of dwellings, such as stables and cowsheds, the prevalence of many species around marshes, and the occurrence of certain "wild" species in forests, would all appear to be due to the fact that in these situations the humidity conditions are relatively favourable to the habits and conducive to the longevity of adult mosquitoes.

(2) The Rôle of Temperature.

Owing to the close relationship existing between temperature and humidity, the following few remarks will be made in regard to the influence of temperature on the life history of *Culex fatigans* and on its power to transmit bird malaria.

The lower limit of temperature compatible with the life of *Culex fatigans* has not been determined, but it is clear from the hibernating habits of these insects that low

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temperature *per se*, if combined with high degrees of relative humidity, is not unfavourable to their prolonged life. At temperatures of about 10° C. (and high degrees of relative humidity) *Culex fatigans* exhibits sluggish movements, it is not readily disturbed, and when disturbed flies slowly, feebly, and almost noiselessly. It also exhibits no desire to feed.

At about 26° C. (and high degrees of relative humidity, but *not* with low degrees of humidity) *Culex fatigans* exhibits marked activity, females usually feed readily, and in fecundated insects (fed on blood) egg development is seen to take place rapidly.

At 40° C. (and high degrees of relative humidity) *Culex fatigans* are killed in about fifteen minutes, in this respect exhibiting a marked contrast to certain species of Indian anophelines, which under the same conditions not only live but exhibit great activity.

In regard to the influence of temperature on the development of *Proteosoma grassii* in *Culex fatigans*, no experiments were carried out, owing to the difficulty experienced in maintaining low mean temperatures in Lahore during April and May.

In the case of human malaria, it is known that the lower limit of temperature necessary for the completion of the sexual cycle of the malaria parasites is a mean temperature of 16° C. (60·8° F. or 289° A.), whilst ROUBAUD has recently shown that the rate of development is in some measure determined by the temperature.

He thus finds that, at a temperature of 25° C., the sexual cycle of benign tertian malaria is completed in eleven days, whilst at a temperature between 17°-20° C. a period of fifteen days is required.

The following observations made at the London School of Tropical Medicine by Colonel ALCOCK (who has kindly permitted me to quote them) and Dr. MANSON-BAHR also point to the same conclusion:—

During July, 1920, 30 *A. maculipennis* were fed on cases of benign tertian malaria and kept under atmospheric conditions. Of these 14 died and 16 survived, and of the latter 11 (68 per cent.) became infected, 4 showing a "large number" of oocysts at the end of eighteen days, whilst 7 showed "a few small oocysts" on the eighteenth and nineteenth days. The salivary glands were negative.

During October, 1920, eight *A. maculipennis* were fed on a case of benign tertian malaria and incubated, in the manner previously described, at a temperature of 22° C. and a relative humidity of 50·8 per cent. Of these, none died and all became infected. Young oocysts were seen on the fifth day, mature oocysts on the eighth day, and sporozoits were found in the salivary glands on the twelvth day. The number of oocysts was in all cases considerable, a stomach examined on the eighth day (when accurate oocysts counts are most readily made) containing over 100.

It is therefore concluded that—

(1) The length of life of mosquitoes, as represented by *Culex fatigans*, is compatible with a wide range of temperature, but at low temperatures their vital functions are restricted.

(2) Temperature exerts a *direct* effect on the malaria parasite in the mosquito, low temperature inhibiting the growth of the oocyst, whilst, within certain limits, its rate of development is dependent upon the mean temperature.

(3) CONCLUSION.

These experiments and observations give rise to the conclusion that the transmission of malaria is dependent upon the coexistence of certain conditions of temperature and humidity, from which it may be inferred that meteorological circumstances, by reason of the combined influence of temperature and humidity—

(1) On the length of life and habits of mosquitoes,

(2) On the development of the malaria parasite in the mosquito,

exercise an important influence in the mechanism of malarial infection.

It is necessary to know the optimum conditions of temperature and humidity for the transmission of the disease, but, whilst these cannot be stated, it has been shown that, in the case of the transmission of *P. vivax* by *A. maculipennis*, a mean

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Zone with mean temperature in July and August :—

Over 62° F. 61° F. 62° F. 60° F. 61° F.

Under 60° F. unshaded.

Recent distribution (1917–19) of endemic malaria = ●

Ancient distribution of endemic malaria ... = ×

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temperature of 22° C. and a mean relative humidity of 50·8 per cent. is highly favourable.

It is of almost equal importance to determine the lower limits of temperature and humidity favourable to the transmission of infection, and these it has been found, by methods fully detailed elsewhere, can be approximately represented by a *monthly* mean temperature of 61° F. and a *monthly* mean humidity (8 a.m. reading) of 63 per cent. It is concluded that when either of the above factors falls below the critical figure "a period of interrupted infection" occurs; and when both remain above the critical figure, "a period of potential infection" ensues.

It will be clear that these factors require to be somewhat delicately adjusted, in order that a period of potential infection should occur. Thus a rise in mean temperature, which otherwise might be favourable to the transmission of malaria, may occasion a fall in relative humidity, which will cause the cessation of infection. Similarly a fall in mean temperature, in spite of a concomitant rise in relative humidity, may have a like effect.

It is thought that considerable importance attaches to the above conclusions, since it enables an explanation to be given of certain points in connection with the endemiology and epidemiology of the disease. For example, it is well known that in the United Kingdom the distribution of anophelines is widespread, whilst indigenous cases of malaria are only met with in certain parts of England, which, as will shortly be shown, are confined to areas exhibiting favourable conditions of temperature and humidity. The absence of any precise relationship between the distribution of anophelines and the distribution of malaria can thus be explained as being due to the fact that the meteorological circumstances favourable to mosquito life are not identical with those required for the spread of malaria.

It is also possible to understand, in the light of the rôle attributed to temperature and humidity, the occurrence in a locality of numerous anophelines and many human carriers, together with a complete absence of new infections (*Paludism sine Malaria*).

On similar lines it is possible to account for the occurrence of new infections—given a period of potential infection—in localities where relatively few anophelines and human carriers are found (*Malaria sine Paludism*)

Time does not permit of the consideration of other points which appear to be explained in the light of the combined influence of temperature and humidity in malaria, but it may be mentioned that evidence has been obtained pointing to the conclusion that the marked influence of rainfall on malaria in the Punjab is mainly the result of its influence on the relative humidity of the atmosphere.

A point of immediate practical importance arising out of the above considerations is the fact that, with a knowledge of the lower limit of temperature and humidity necessary to create a period of potential infection, it is possible, in the case of any area for which the necessary meteorological data are available, to determine the season of the year and the precise duration of the period during which malaria may be acquired. In the Punjab for example it is found that malaria can normally only be acquired during the months of March, July, August and September (sometimes also in October and November), whilst in England, in normal years, the transmission of infection is limited to the months of July and August. An examination of the meteorological data of the United Kingdom also shows that in a large part of England and Wales, the whole of Scotland and the whole of Ireland, the temperature conditions are normally unfavourable to the transmission of malaria, in which areas it would therefore appear that anti-mosquito measures, as a means of preventing the possible spread of malaria, are not a matter of the first importance.

In conclusion, I desire to state that, whilst undue importance is not attached to the results of what is necessarily an incomplete and preliminary investigation, it seems a justifiable assumption, from the nature of the evidence briefly indicated in this paper, that the rôle of meteorology in malaria is a subject of considerable importance. In these circumstances the view that meteorological conditions mainly favour malaria through their influence in creating innumerable breeding places for mosquitoes appears to be no longer tenable. May it not be that there are more things in *heaven* and *earth* affecting the malaria problem than are dreamt of in this philosophy.?

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Discussion.

Dr. Angus Macdonald: I have listened with great interest and pleasure to the paper read by Colonel Gill, and think this Society is honoured by the communication. The value of his experiments and observations lies in that he is breaking new ground—new ground, not in the sense of recognising relation of temperature and humidity with malaria and mosquitoes, but in the recognition of their significance in protozoal development and infection, and hence in prevention. Humidity and temperature *have* been considered by the authorities on malaria, but not till recently with pertinence to their specific epidemiological aspects, which became possible only after the discovery of the plasmodial cycle within the mosquito.

I have learned much from Colonel Gill's observations on the effect of humidity on mosquito life and plasmodial sporogeny. Having during recent years studied the relation of temperature to malaria in this country, where the relative humidity is comparatively high and the range narrow, and had the opportunity of reading the war-time reports from Mesopotamia and other fields, where range of humidity is wide and a low degree is reached, I have felt satisfied of the epidemiological negligibility of humidity. In this country, in the brief period of normal years over which temperature is sufficiently high to produce infection, the degree of humidity reached seems to effect no change in the life and habits of the anophelinæ. It is, of course, at lowest, well above the critical point named by Colonel Gill. In Mesopotamia the record of malaria "admissions" holds an even course throughout the months of low humidity, and shows definite reduction only in the winter months of low temperature. From reports, however, I find indication of reduction in malaria infection during the months of highest temperature. Some gross fluctuations might be expected if humidity were of epidemiological significance, and would be evident in Mesopotamia if anywhere.

There are many conceivable ways—and I emphasise this to deprecate any generalisation from laboratory experimentation—there are many conceivable ways in the habits of the anophelinæ whereby a natural evasion of desiccation may have come about. The observations of Colonel Gill on *Culex fatigans* in Lahore point to one manner of such evasion: the mosquitoes dead out of doors, exposed, no doubt, to sun heat as well as to low humidity, and alive within at shade temperature and higher humidity. Colonel Gill does not give the temperature differences in this observation nor in the consideration of feeding irregularities; temperature alone might account for these happenings, though that a certain mean of humidity is demanded for the continuance of existence may be taken as a physiological axiom. Warmth and moisture, to put it that way, are essential for the life of creatures. Absolutely, therefore, humidity and temperature are of equal importance. Relatively, however, I should say *the epidemiological influence of temperature is universal; of humidity, local.*

It may be granted, however, that data, experimental and epidemiological, are lacking. A wide accumulation of observations in nature is needed; experiment aids the explanation of the occurrences. Administration should be able to obtain pertinent records from India, Mesopotamia, the oases of Africa and Arabia, from the Straits Settlements and elsewhere; situations with wide variations in rainfall, temperature and humidity—relative humidity of below 30 per cent. for three or four months of the year round Babylon and of over 90 per cent. at Malacca. As regards malaria, the first essential is the accurate collection of the facts of incidence with correct parasite diagnosis and the differentiation of "primary" and "relapse." In the laboratory the maximum, minimum and optimum temperature for sporogeny may be ascertained; and these facts should be regarded in no absolute sense, but used with caution in harmonising them with epidemiological observations. The knowledge sought of the significance of both factors is essential for preventive purposes, and, as I have elsewhere stated, I can conceive a revolution in the *economy* of prevention in the acquisition of that knowledge.

In my work I have stressed "temperature." Colonel Gill entitles his paper "Humidity." He, however, seems to progress towards the emphasis of temperature, and may probably find "Temperature and Humidity" more apt.

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Dr. B. BLACKLOCK: One thing which appears to me possible to criticise is that, in certain particulars, Colonel GILL's general conclusions are slightly in advance of the results obtained by him in his experimental work. He has mentioned "paludism without malaria." In this connection he says, "It is also possible to understand, in the light of the rôle attributed to temperature and humidity, the occurrence in a locality of numerous anophelines and many human carriers, together with a complete absence of new infections (paludism without malaria)." The author, in his experiments, has proved two points: he has proved that if you reduce the relative humidity to a point below 45 *Culex fatigans* dies. Thus you have an interruption in the transmission of *Proteosoma grassii* from one bird to another. The second point which he has proved is that, given the survival of the *C. fatigans*, the infection, rate of development, and completion of development of *P. grassii* go on in a constant manner.

Take the first point, namely, the death of *C. fatigans* at a low humidity; if we reason on analogy towards Anopheles, we might conclude we should find a relative humidity below which anophelines would die, thereby interrupting the transmission of human malaria. Such an explanation might be possible in the case of the experiment quoted by the author, by ALCOCK and MANSON-BAHR, in which fourteen anophelines died. But if that were the explanation of their death, which would be analogous with the effect on *C. fatigans*, it is clearly not the explanation of "paludism without malaria," of which an essential factor is, not the death of anophelines but their existence in very considerable numbers

Another point he has proved is the constant development of *P. grassii* in *C. fatigans*, provided it escapes the effect of low relative humidity. If, therefore, we find that sixteen anopheles in the experiment survive, and that the rate of infection and the rate of development of the malaria parasites in those anopheles, and, finally, the completion of the cycle, are adversely affected, it is clear that we must not assume that this is the result of any variation in the relative humidity, which, on the author's own showing, has no such effect in his own experiments.

The figure 63 is stated to be the critical relative humidity figure. I do not know that I have the whole of the information here at my disposal, but I think it means that, as regards anophelines, below that figure there are certain factors at work, depending on relative humidity, which interfere with the transmission of human malaria. I do not think that in either the death of the mosquito or the retardation of the cycle one can attribute this effect to relative humidity. And, moreover, I cannot explain the figure 63 when I see that, in the experiment quoted as No. 2 of ALCOCK and BAHR, a relative humidity of 50 was found to be highly favourable.

The statement of the author's observations in connection with the number of oocysts which develop in proportion to the number of parasites existing in the blood of the birds, is not, I think, quite borne out by certain experiments in malarial transmission, nor, I think, in trypanosome infections, as shown by the work of Miss ROBERTSON, on the effect of trying to infect Glossina where parasites are numerous or otherwise in the blood of the mammal host.

The question of carrying out anti-malarial measures seems to me a rather large one to introduce at this stage of the research. Colonel GILL's valuable experiments will, no doubt, be repeated many times, and will in time give us some definite guide. It is also true that in several countries, up to the present, without this knowledge, malaria has in certain localities diminished very considerably; but I think it would be a little premature to suppose that the influence of variation in relative humidity was the most important or even one of the most important factors in causing this diminution in malaria and its elimination.

Dr. P. MANSON-BAHR: I did not intend to take part in the discussion, but I must explain a few points about the experiments made during the past summer by Colonel ALCOCK and myself. The unfortunate death of fourteen anopheles, which Dr. BLACKLOCK has referred to, can hardly be ascribed to the effects of temperature and humidity, but rather, I should say, to the effects of August Bank Holiday. It was during this period that

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a great increase in the death-rate of these insects occurred, and was probably due, to a great extent, to hunger and thirst, as the mosquitoes were not fed so regularly on glucose solution during the holiday period.

I am sure Colonel GILL, in his paper, did not intend to attribute any meaning to these figures; we had not up to that time had the advantage of his co-operation. One learns only by experience, and I very much hope, as the result of Colonel GILL's work, we shall be able to get better results during the coming summer.

Dr. C. M. WENYON: From what I understand Colonel GILL to mean, his experiments demonstrate that when mosquitoes are made to live in atmosphere with the humidity below a certain level they shrivel up and die—in other words, mosquitoes cannot withstand drying. It has not been proved that humidity *per se* has any effect on the development of the malarial parasites, for the mosquitoes died when exposed to low humidity before the development could take place. Temperature, however, affects the rate of development in the mosquito.

I do not know how the mosquitoes were fed in the experiments, but, from my own experience, the life of the mosquito depends on several factors. Mosquitoes when kept too moist often swell up, and are very liable to a bacterial infection which disorganises the intestine and leads to their death. On the other hand, if they are too dry they shrivel up and die. If, however, mosquitoes are kept in a dry atmosphere, and with arrangements for giving them access to moisture, they live very well. In Bagdad, were the atmosphere is very dry and the summer temperature up to 110°–120°, the mosquitoes have the habit of sitting on the outside of the large porous native earthenware filters, where they not only obtain moisture but are kept cool. During this time the humidity of the atmosphere is very low, whereas in close contact with the filter, I imagine, it would be much higher. These conditions are difficult to repeat in an experiment in comparatively small enclosures. We would have to arrange for the continuous circulation of air of definite humidity through the enclosure, within which should be an imitation of the native filter, to which the mosquitoes could repair if they desired. As Dr. MACDONALD has stated, the native mosquitoes have many ways of avoiding the dry parts of the atmosphere.

Colonel GILL has told us that when the humidity of the atmosphere to which mosquitoes are exposed falls below a certain limit they die, and if this takes place in nature the chances of spread of malaria are lessened.

Mr. A. BACOT: I would like to put this question: What means were taken to ensure circulation of air in the incubator? A correct reading of the hygrometer cannot be obtained in a still atmosphere. I did not understand from Colonel GILL, in the case of the mosquitoes which died in seven days, if they were fed. Perhaps he will tell us in his reply.

With regard to Dr. WENYON's remarks, if the mosquitoes did not go to the native filters, or had not some other cool situation to retire to, they must be killed. An air temperature of 120° F. (45·7° C.) would be sufficient to kill them, not by drying them up but owing to its effect on the nervous system. Insects die of a kind of heat stroke between 45° and 52° C. In an incubator insects remain active up to a certain temperature; but a couple of degrees higher will result in their death within five minutes. It is not a question of drought; they are quite flaccid after death.

I would like to refer to the work concerning isothermic conditions in this country. Work in this connection is very interesting to entomologists, and valuable even when done on the large scale lines put before us to-night, but to ascertain the accurate effect in regard to insect life, it is necessary to have more minute details with regard to both temperature and humidity. In this country the more important factor is temperature. In the Fens of Cambridgeshire there is a low local temperature over dark soil amid the tall reeds, which differs from that on the bank of the dyke alongside the fen. This localisation also applies to the Essex marshes near the mouth of the Thames. It may be that the heat absorbed by the dark soil constitutes a reserve which makes all the difference to insect life. On the Essex Saltings certain insects are found which, though commonly distributed in Switzerland and other parts of the Continent, occur only on the mud

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flats of the East Coast of England. This has been shown by entomologists to be connected with isothermic conditions.

Sir RONALD ROSS: In regard to the feeding experiments, I would like to ask what these insects were fed on. I may add that a large number of researches has been done on this subject, and remember that about eighteen years ago an Austrian or Hungarian, N. JANCSÓ, conducted a number of researches on the effect of temperature on the growth of parasites and mosquitoes of various kinds. I do not know whether he did any work on humidity. Mr. LE PRINCE, of Panama, did a good deal of work on the effects of temperature, and also, I think, on humidity.

I have very little more to say, except that everyone has recognised the effect of humidity and temperature on mosquitoes, though we have not made such particular researches as has Colonel GILL. For instance, I remember that on several occasions my mosquitoes, which were nicely infected, perished simultaneously because, by accident, they had been left out on the mosquito nets in the verandah. Another experience was that, as Dr. WENYON has said, mosquitoes go to the water-jars to keep damp. We also know, by painful experience, how much mosquitoes are apt to bite in damp warm weather. Nevertheless, I like to know that Colonel GILL's definite experiments have been carried on.

One point I wish to emphasise is this: malaria is a function of about fourteen variables; yet medical men seem to think that only one variable is concerned in it. I have often written on this. There are about fourteen variables on which the amount of malaria in a given place must depend. You cannot judge properly by considering one or two. Temperature is one, humidity is another; all must be considered. You must not come to conclusions hurriedly, as this paper shows some tendency to do towards the end.

Lieut.-Colonel C. A. GILL: In answer to Sir RONALD ROSS, the mosquitoes were fed during the course of the experiments on raisins, which were placed in mosquito "hotels," one of which may be seen inside the incubator. When low degrees of relative humidity were being used we also deliberately watered the mosquitoes once a day, by means of a wet sponge placed on the gauze covering the mouth of the "hotel." They were often seen to fly up and insert their probosces through the gauze into the sponge and take a deliberate drink. This also answers the question asked by Mr. BACOT.

With regard to the fourteen "variables" mentioned by Sir RONALD ROSS, it is recognised that other important factors are concerned in the mechanism of malaria, although in this paper an attempt has only been made to deal partially with two of them, the importance of which, I think, has not been fully recognised and appreciated. I am conscious of the justice of the criticism that my conclusions appear to be somewhat wide, but I should like to emphasise the fact that they are founded on an investigation only a small part of which has been presented to-night. So I hope my error in this respect is not actually so great as it may now appear.

In reply to Dr. BLACKLOCK, I fear I did not make myself clear in regard to the critical humidity being 63 per cent. I said I found, by a method not described, that a monthly mean humidity of 63 per cent. (8 a.m. reading) was approximately the lower limit of relative humidity necessary for the transmission of malaria. The whole point is the 8 a.m. reading.

The isopleths of humidity, which I exhibited on the screen, showed that there is an extremely marked diurnal variation in humidity. In the case of Kew, for example, it declines by about 20 per cent. between 8 a m. and 10 a.m. There is, therefore, nothing inconsistent between a relative humidity of 63 per cent. (8 a.m. reading) and a mean relative humidity during the twenty-four hours of 50·8 per cent. They, in fact, correspond fairly closely in the Punjab. With regard to the conclusion that the temperature and humidity factors may sometimes be responsible for the condition known as "paludism *sine* malaria," what I meant to imply was that, if the mean temperature or the mean humidity was below the critical figures, new infections with malaria would not take place in spite of the presence of numerous anophelines and human carriers. The British Isles is an example of an area where we know anophelines abound, and numerous human

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carriers have recently been imported, yet indigenous cases of malaria are, it has been shown, confined to relatively small areas where the temperature is favourable. The rest of the United Kingdom, therefore, affords an example of paludism *sine* malaria, due for the most part to unfavourable conditions of temperature.

Again, in Lahore, in May, adult *C. fatigans*, which were breeding out daily in vast numbers, could be readily captured, as well as sparrows infected with Proteosoma; yet, owing to the extremely low relative humidity, it is thought (in view of the experimental results) that fresh infections could not take place, owing to the fact that no infected insects would survive long enough for sporozoits to appear in their salivary glands.

In this paper I have rather emphasised the temperature factor, because I have attempted to apply the rôle attributed to temperature and humidity in determining the distribution of malaria in England. Under English conditions the temperature is in the vicinity of the critical temperature, whereas the humidity factor is invariably high and well above the critical figure. Thus, in this country, of the two factors under consideration, the more important factor is that of temperature, whereas in tropical countries, where the temperature factor is usually favourable, the humidity factor is subject to wide variations, and is, therefore, the more important.

Dr. WENYON mentioned the humidity of Bagdad. I do not know the figures, but at Babylon the mean relative humidity, as measured at 7 a.m., 2 p.m. and 9 p.m., is below 34 per cent. from March to October inclusive, whereas the mean relative humidity at Richmond, in England, measured in the same manner, is never less than 67 per cent., and usually considerably higher.

In a country such as Mesopotamia, therefore, where the mean relative humidity is often for long periods as low as 20 per cent., local conditions, such as marshes or proximity to rivers, which occasion *locally* a high mean relative humidity, will be likely to affect the amount of malaria. Mosquitoes will be able to thrive and survive in these situations, and it, therefore, seems permissible to assume that in tropical countries with a dry climate, local variations in the incidence of humidity will be in some measure responsible for local variations in the incidence of malaria.

With regard to the point that the degree of infection of *C. fatigans* was found to accord closely with the number of parasites present in the blood of the sparrow, as determined on the day of feeding, I am aware of the contrary conclusions of SERGENT, and I can only say that when we used a sparrow for feeding purposes whose blood had not been examined for about a week, we usually got little or no infection of the mosquito, whereas when a parasite count was made on the day of feeding, there was a distinct relationship between the number of parasites in the blood film and the number of oocysts which subsequently developed in the midgut of the mosquito.

Indeed, by adopting this precaution, and by incubating the mosquitoes under favourable conditions of temperature and humidity, in the manner described in this paper, the carrying out of transmission experiments in the laboratory became a matter of such ease and certainty as to suggest that the principles underlying the method may prove of value, not only in investigating the transmission of insect-borne diseases in general, but also, in certain cases, as an aid to diagnosis and as a check on treatment.

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THE USE OF THE SIGMOIDOSCOPE AS AN AID TO DIAGNOSIS IN CHRONIC DYSENTERY.

BY

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AND

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The correct diagnosis of relapsing dysentery, or of chronic diarrhœa, amongst war pensioners, is a matter of very considerable importance from an individual, as well as from a national point of view. Considerable difficulty may be experienced in making a diagnosis by a simple microscopical examination of the fæces in men who contracted their dysentery three or four years ago, and it will be our object to point out how this difficulty may, to a great extent, be overcome.

The chronicity of these cases, as well as their tropical origin, would at first sight suggest that the majority are amœbic, and this statement, to a great extent, proves to be correct; but it by no means follows that all are due to the *Entamœba histolytica*. Quite a number are not, and these non-amœbic diarrhœas are by no means improved—it may be that their symptoms are aggravated—by a misdirected anti-amœbic treatment.

Sometimes little assistance may be gained from a consideration of the patient's history alone, for it is not exceptional to encounter cases in whom the primary diagnosis during active service was bacillary dysentery, but who in their subsequent military career contracted amœbiasis. Mere physical examination of the patient can provide little definite assistance.

It is true that, as a rule, chronic bacillary cases, whose mucosa has been to a considerable extent destroyed, are more emaciated and bed-ridden than are the chronic amœbic ones.

Palpation of the abdomen in the former disease may fail to reveal any localised tender spots, whereas amœbic cases more usually exhibit areas of deep tenderness situated over the course of the large intestine, especially the cæcum and transverse colon.

We wish to emphasise that in the bacillary disease it is the terminal portion of the large gut which is most seriously affected, a circumstance which renders sigmoidoscopic examination so valuable in this condition, especially as a positive diagnosis cannot be obtained by any other method. We wish to explain briefly why this is so.

The pathological changes present are due to fibrosis and the deposition of granulomatous tissue—the aftermath of the acute bacillary disease. Therefore a simple microscopic examination of the fæces is of a purely negative value, nor can we expect to obtain much assistance from cultures for the dysentery bacilli, as these have long disappeared. Serological reactions also have long ceased to be of any diagnostic importance in these cases.

It is well known that in amœbiasis an amœbic carrier may pass numerous cysts of *E. histolytica* on one or two occasions, but that subsequent examinations—it may be for days, or even weeks on end—may fail to reveal any traces of this organism. We may instance one particular case, in which eleven fæcal specimens were examined, with a

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negative result, but on the twelfth occasion numerous characteristic cysts were seen; this occurred in a patient who was being actively purged, and was passing stools containing mucus and blood at the time.

One is justified in assuming that cases of this nature are by no means exceptional.

There are also cases—three of which have recently been encountered—in which active entamœbæ were found in the blood-stained mucus passed during a brief exacerbation, but who subsequently continued to void fæces containing neither the vegetative forms nor their cysts. Microscopic examination of the fæces for the *E. histolytica*, or its cysts, is a very necessary procedure, and we do not wish to make any statements which would bring such a well-established method into disrepute, but it has its limitations, as every experienced microscopist will admit.

It is now clear that, as a routine method of excluding an amœbic infection, many microscopical stool examinations are required for each patient, and, incidentally, many days must elapse before a definite diagnosis can be made or a rational treatment instituted. Any system, then, which tends to obviate this delay is to be encouraged as saving both time and money.

During the past year, we have employed the sigmoidoscope as a means of assisting diagnosis and in controlling treatment, and by this means, when used as a routine in chronic dysentery, we think that we can overcome the difficulties we have detailed and shorten the patient's stay in hospital, and thereby save the State considerable expense.

The advantages of the routine use of the sigmoidoscope are:—

(1) A rapid and accurate diagnosis by direct vision can often be made.

(2) Unsuspected intestinal conditions, such as hæmorrhoids, syphilitic stricture, malignant disease or polypi are occasionally revealed.

(3) The proportion of cases of doubtful etiology is considerably reduced.

The method adopted in making a sigmoidoscopic examination is comparatively simple, but it is necessary to emphasise one or two points.

The preparation of the patient for rectal examination requires consideration. He should be purged so that the large bowel is emptied of fæces, but at the same time it must not be rendered too irritable.

We have found that the most satisfactory method consists in administering half an ounce of castor oil on the previous evening, followed in the morning by a soap and water enema so as to completely clear out the rectum.

In order to render the bowel less sensitive to manipulation, and to check peristalsis, we find it advisable to administer 15 min. of *Tinct. opii* immediately after the enema.

In cases where there is incessant diarrhœa a satisfactory method of clearing out the bowel has yet to be discovered, but, fortunately, in these cases a fleeting glimpse of a small portion of the bowel usually suffices to establish a diagnosis.

As regards the posture assumed by the patient during examination, we prefer the lithotomy position. This position causes any fluid contents of the bowel to collect in the dorsal aspect of the rectum, which acts as a sort of a trap, while the bowel, both above and below this pool, may be minutely scrutinised.

It is quite unnecessary, indeed to our minds it is inadvisable, to administer an anæsthetic. We should be guided by the patient's sensations of pain and discomfort while the instrument is being passed.

Naturally, when one intends to examine an ulcerated bowel, great care should be taken in introducing the instrument, and when once the sphincter muscles have been passed its further passage should be guided by sight alone.

The degree of pain experienced varies greatly. Generally it is much greater in the bacillary types than in the amœbic disease; it is much greater in highly-strung and nervous individuals than in less sensitive subjects. The ulceration of the bowel itself would seem to have little bearing upon this point, for some cases with extensive ulceration complain of little discomfort whilst being examined.

It is wise not to disregard any sudden pain of which the patient complains, for this is generally due to too great pressure being exercised upon one particular portion of the bowel wall.

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The distance to which the instrument can be passed also varies considerably; in some a length of only four inches, in others, again, twelve inches or so, may be reached with ease, till the point of the sigmoidoscope can be distinctly felt in the left hypochondrium.

Appearances of Amœbic Ulceration.

The following lesions can be made out:—Discrete diamond-shaped ulcers of varying size and extent with hæmorrhagic margins and greenish or grey basal sloughs. Some appear to be quite shallow, others of some considerable depth. The intervening mucous membrane is healthy in appearance and yellowish-pink in colour, though generally it is more redundant than is the normal. On the whole, the lesions resemble those seen at autopsy, but it has struck us that the small petechial hæmorrhages which are obscured after death and the general vivid colouration of the blood clots which cover the smaller ulcers are much more striking than when seen in the dead body.

This is what one would expect from a study of living tissues.

In a small percentage of undoubted, that is, microscopically diagnosed amœbic cases, the rectum is not ulcerated at all; in these instances it is probable that the amœbic lesions are situated in the upper part of the large bowel beyond the range of vision through the sigmoidoscope, and the stools in these cases usually contain *E. histolytica* cysts, rather than vegetative entamœbæ.

Important as are the sigmoidoscopic appearances, much information may be obtained from microscopical examinations of the scrapings of these ulcers.

The best instrument for this purpose is a wooden match affixed to a holder and introduced through the sigmoidoscope. By this means individual ulcers may be scraped and the exudate derived from them microscopically examined. It is possible by this method to demonstrate active entamœbæ in considerable numbers together with Charcot-Leyden crystals, which themselves are possibly diagnostic of amœbiasis (J. Gordon Thomson) in cases in which several fæces-examinations have proved to be negative.

Sigmoidoscopic Appearances in the Bacillary Cases.

The lesions seen in the lower bowel in this very serious condition consist for the most part of granulation tissue, and represent an attempt on the part of the bowel to replace, as far as possible, the tissues originally destroyed by the primary specific necrosis. Inflammatory changes, when present, are due probably to a secondary invasion of the badly-lacerated mucosa by intestinal organisms other than the dysentery bacillus.

We have not seen cases where *ulceration* of the bacillary type was actually present in the rectum. The changes we have seen can be classified, according to their severity, as follows:—

(1) The mildest form appears as a slight hyperæmia of the mucosa, which is more sensitive than usual to the passage of the instrument.

(2) Acute hyperæmia of the mucosa, together with a slightly granular and glazed appearance throughout the rectal canal, or, it may be, occupying definite zones, three or four inches in diameter.

(3) Masses of granulation tissue superimposed upon an atrophic and fibrotic mucous membrane.

(4) Granulomatous polypi arising from a hyperæmic or hæmorrhagic bowel wall. This condition may extend throughout the large intestine, for similar changes have been found in the cæcum of two cases at operation.

Cases with lesions of this extreme severity are usually in a critical condition.

We append statistics to illustrate the points alluded to in this paper, and we wish to thank Dr. G. C. Low for so kindly permitting us to utilise figures obtained from cases under his care, as well as permitting us to exhibit drawings made from one of his patients.

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DIAGNOSES IN ONE HUNDRED CONSECUTIVE CASES OF SUSPECTED DYSENTERY.

By Sigmoidoscope.

Positive amœbic	29	42
Suggestive „	13	
Positive bacillary	15	28
Suggestive „	13	
Other causes of symptoms:—		
Rectal polypus	1	
Hæmorrhoids	4	
Syphilitic stricture	1	
Etiology undiagnosed	11	
Normal appearance	14	
	*101	

*The extra number is explained by the diagnosis of amœbic and bacillary infection in the same patient.

By Microscope.

Positive amœbic (Charcot-Leyden crystals, etc.)	30	31
Suggestive amœbic	1	
Positive bacillary	0	5
Suggestive „	5	

Remaining of little positive value ... ... 211 exams.

Total microscopical exams. done on these 100 cases ... 247

Only two cultures were made—both negative.

These preliminary examinations were all performed before a diagnosis was established.

Details of Sigmoidoscope Results.

44 positive diagnoses were made, and of these—

20 were confirmed by the microscope.
15 others were confirmed by the clinical history of the patient.
In 8 the clinical history was doubtful, and confirmation of the diagnosis is lacking.
In 1 the history disagreed with the diagnosis, which was proved clinically to be correct.

26 suggested diagnoses were given, and of these—

6 were confirmed by the microscope.
10 others were confirmed by the clinical history.
In 5 the clinical history was doubtful, and confirmation of the diagnosis is lacking.
In 5 our diagnosis was probably wrong.

Thus there were 52 confirmed diagnoses of amœbic or bacillary dysentery, and there were 6 cases in which the symptoms were caused by other local conditions, such as hæmorrhoids, etc.

Of the remaining 43 findings—

11 were undiagnosed.

14 appeared normal, and of these—

6 had no symptoms, and were discharged.
1 had *E. histolytica* cysts and a liver abscess, but had never had dysentery.
2 had *E. histolytica* cysts in their stools.
3 gave a history of amœbic dysentery.
1 gave a history of bacillary dysentery.
In 1 the clinical history was doubtful.

13 lack confirmation.

In 5 our diagnosis was probably wrong.

In other words the sigmoidoscope was instrumental in giving a positive diagnosis, that is, a confirmed diagnosis, in 58 per cent., while in the same hundred cases the microscope alone gave 31 per cent.

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RICKETTSIA IN THE BED-BUG: DEMONSTRATION.

Mr. A. BACOT gave a demonstration showing a species of Rickettsia in the bed-bug which, although it has probably been noticed before, in one or other of its stages, has not apparently received serious attention or comment. Rickettsia bodies are a new group. It is now generally admitted that they are organisms showing many resemblances to bacteria in their staining reaction and morphology, but, in the absence of definite cultural successes, it is impossible to say how nearly related to bacteria they may be. They are usually diplococcoid in form, and have staining capacities in common. Rickettsia bodies are associated with Rocky Mountain spotted fever, conveyed by ticks, trench fever, and typhus fever conveyed by the body louse, and, I think, Japanese river fever conveyed by mites. Some persons consider them to be the causal organisms of these diseases; others think they are not associated with the disease itself but only with the insect conveying the disease. The species which my colleagues, Dr. ATKIN and Dr. ARKWRIGHT, and myself found in the bed-bug exhibits considerable similarities in its development to *Rickettsia prowazeki*, the supposed causal organism of typhus fever. It is intracellular, and in addition to the diplococcoid form shows both bacterial and thread-like phases. These latter forms are not seen in the Rickettsia associated with trench fever. In other respects, however, the species in the bed-bug is more akin to that which NÖLLER described as occurring in the "sheep ked" *(Melophagus ovinus)*. It passes from generation to generation through the egg, and, so far as is known, has no second host. The eggs become infected before they are laid. In the intracellular stage clusters occur in the cytoplasm of the cell. A smear of the ovary shows both minute pointed-ended forms together with developing bacillary forms. Examples of the long bacillary forms are also shown under one of the microscopes. In certain cells of the Malpighian tubules the development is very pronounced, the cell becoming greatly enlarged; it may be two or even three times the diameter of the normal tube. If these enlarged cells are broken or cut across, the interior is found to be filled with a mass of long thread forms, and sometimes a considerable proportion of the minute coccoid bodies are present as well.

Apparently all bed-bugs are infected, and this is thought to be also the case with the "sheep ked."

I would like to ask any members who come across the species of Cimex which infest the haunts of pigeons or bats, kindly to communicate with Dr. ATKIN or myself, as we are anxious to ascertain if the bugs of animals other than man are infected.

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TRANSACTIONS

OF THE

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Proceedings of a Meeting of the Society held on Friday, February 18th, 1921, at 8.45 p.m., at 11, Chandos Street, Cavendish Square, W. 1., Professor W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P. (*President*), in the Chair.

FIVE CASES OF FASCIOLOPSIS INFECTION, WITH REMARKS.

BY

A. F. COLE, F.R.C.S. (ED.), FORMERLY OF NINGPO, CHINA.

The rarity of such infections is obvious from a study of the literature; the existence of an endemic focus where material for clinical and pathological study is always available is not generally known.

Although about one hundred miles from the district in point, a large number of patients admitted to the wards of the C.M.S. Hospital, Ningpo, from Shao-hying-fu, were infected, and the notes on five cases appended by no means represent all the cases seen.

As regards the histology of the flukes, I regret to say that of the many hundreds of sections made by me in attempting to differentiate the species, I have lost every one during the war. From my study of the sections—longitudinal, transverse and horizontal—I did not observe any differences of moment between the so-called *F. buskii*, *rathouisi* and *goddardi* types.

A well illustrated and most thorough paper by N. W. BROWN, "Fasciolopsinæ of China," *Bulletin Johns Hopkins Hospital*, Vol. 28, pp. 322-329, goes into the question of cuticular spines, opercula, variations in size of ovum and parasite; and J. W. W. STEPHENS, after describing the Fasciolopsis group in *Animal Parasites of Man*, remarks, in conclusion: "It is evident that a re-examination of fresh material is required before the validity of all these species can be accepted."

It is with some diffidence that one attempts to add anything to N. W. BROWN'S

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monograph on Chinese Fasciolopsinæ, but having had the privilege of examining some hundreds of *fresh* flukes—in many cases alive and squirming like a freshly caught flatfish—I wish to suggest that the differences in measurement of length and width and thickness are compromised by the fact that a dead fluke when passed may be in any stage of degeneration, from mere muscular relaxation to actual commencing maceration and digestion. Kwan's fluke (so-called) illustrates how a new species may be discovered; by inspection of the original specimen in the helminth collection at Endsleigh Gardens Tropical Diseases Hospital, it is obvious that it has no claim on recognition as anything more than a macerated *F. buskii*. Scores of similar specimens were noted whilst investigating case 729.

The freshly passed flukes appear reddish brown, like raw beef, with marked movements in many cases. They did not alter position in the fæcal pot, but I have no doubt they do so within the bowel; the cuticular spines and the musculature would conceivably act to this end, and the epigastric discomfort and irregular diarrhœa, commonly complaints in these cases, might be caused by migratory movements. Whilst on the subject of movement, I may say that I have no recollection of having seen any specimen of N. W. BROWN'S Group I. *un*spined Fasciolopsinæ in actual motion. This may point to the unspined feature being merely a post-mortem product; if so, the differentiation into *F. buskii* and *F. spinifera* becomes useless. Elongation, flattening, visibility of the vitellaria, as seen by naked eye in freshly passed specimens, may be explained by the same post-mortem action, and may account for discrepancies in the records of various observers of the spines in this group. The ova illustrated by Dr. N. W. BROWN may also point in the same direction—the contents degenerating and the opercula tending to be missing.

I have endeavoured to avoid the question of identification of species and have failed. As a result of my own observations, I incline to the belief that in the part of Che-kiang province where I have lived, it has not been clearly shown that there is more than one species, *Fasciolopsis buskii*. As regards clinical facts, the cases appended suggest:—

(1) The parasites live in the stomach, duodenum and the first part of the small intestine only; when found in the large bowel they are nearing the end of their existence.

(2) Their ill effects are not in the direction of blood destruction, but are produced by toxic absorption, which leads to secondary anæmia, dyspnœa, general œdema, ascites, and skin changes. There is no fever directly caused by them.

(3) No melæna has been noted, even in the massive infection case 729. Diarrhœa is common. Many of the patients state they have passed blood, but a freshly passed fluke in a mass of pultaceous fæces exactly simulates blood clot; moreover, there is a Chinese proverb: "Ten men, nine hæmorrhoids; Ten women, nine leucorrhœas"; which bears on the subject of melæna.

(4) No cases were observed in infants or young children; this may be connected with the fact that weaning is often delayed to the third and fourth year.

(5) The disease may be fatal unless the parasites are expelled by art or by accident. I had one death (not here recorded) within twenty-four hours of admission for bowel troubles, in which the typical ova were demonstrated before death.

(6) Rapid recovery follows when the parasites are evacuated, unless the patient is in the later stages of exhaustion.

(7) Fæcal examination and re-examination are the essentials for success, the fasciolopsis ova being characteristic. Certain observations were made on the relative sizes of ova *in utero* and in the fæces; also in the analogous case of *Fasciola hepatica* infection in the pig of Shanghai abattoirs.

CASE 502. Case under Dr. A. F. COLE. September, 1908.

Ningpo boy, æt. 15, quite well in Ningpo. Went to work as apprentice to carpenter in Siao-saen, near Shao-hying-fu, Chekiang. After six months there developed symptoms which brought him to Hospital in Ningpo.

On admission he was unable to stand from weakness, lies in an extremely dyspnœic condition, and there is advanced general œdema. No history of melæna (no note made of presence or absence of diarrhœa.—A. F. C.).

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History of having vomited flukes, and fæces contain certain ova averaging 135μ × 85μ.

Large doses of male-fern extract no effect; three doses of thymol, amounting to 45 grains, on an empty stomach produced 113 flukes per anum. Patient insisted that he could hear the flukes crying out before they were passed! Measurements of flukes from 29 to 42 mm. in length by 9 mm. to 16 wide.

CASE 519. Case under Dr. A. F. COLE. July, 1910.

Servant of Rev. H. Barton, C.M.S., Shao-hying-fu, Chekiang.

Complains of great weakness, and irregular diarrhœal attacks without any blood. Appetite fair, not feverish, tongue is coated, great discomfort in epigastrium. No œdema present. Wasted appearance.

Examination of fæces on three days, some 20 slides in all, gave in addition to ova of *Trichocephalus dispar*, five oval single contour granular ova of following measurements:—

$$\left.\begin{array}{l} 170\mu \times 85\mu \\ 170\mu \times 85\mu \\ 200\mu \times 90\mu \\ 180\mu \times 80\mu \\ 170\mu \times 95\mu \end{array}\right\} \text{Mean} = 178\mu \times 87\mu.$$

Eucalyptus oil ʒi with chloroform ʒs, and oleum ricini ℥ss was given, and five hours later he was said to be in agony, which was relieved by vomiting. In spite of the strictest orders his motions and vomit were thrown away into the common lot, and, though search was made, no discovery of flukes. In January, 1911, the patient says he has been in good health ever since the above treatment; living now in Ningpo.

CASE 606. Under care of Dr. A. C. HUTCHESON, Kashing, Chekiang. June, 1911.

Slave girl, æt. 7, from Siao-saen, near Shao-hying-fu, walked into Dispensary with five flukes on a mulberry leaf, which flukes she had vomited. History of having both vomited and passed flukes per anum on several occasions. Child is pale and weak, looks very sick, motions very loose and frequent, and ova of liver flukes present, average of 20 ova = 143μ × 82μ.

Thymol grs. xii. on three occasions brought away some fifty flukes. Child left Hospital in fine condition, improving rapidly.

CASE 729. Under care of Dr. A. F. COLE. April, 1911.

Schoolboy, æt. 17, Hangchow bred, from C.M.S. Trinity College, Ningpo, admitted for wasting and general debility. Commenced with mild diarrhœa 2–3 times a day about 2½ months ago, about the same time that he was in Shao-hying-fu, but he says he was there only two days and already unwell. He says he never has passed blood; he is too weak to play any games, or even to hold a tennis racquet firmly. He has a mild diarrhœa of a chronic nature.

Present condition.—Patient looks wasted and seems very weak; skin and face dry and scaly. Pulse 72. Respiration normal, Temp. normal. Breathlessness on exertion. Urine 1010, acid, no albumen, reduced Fehling's solution on one occasion (?). Lungs normal. Heart, all 2 sounds accentuated, no murmurs. Discomfort in epigastrium. Fæces said by dresser to contain only Ascaris ova. Blood contains no parasites: reds 3,443,000, whites 8,000, hæmoglobin 90% (differential white count not made). About 30–40 ounces of urine passed in 24 hours. There is no œdema and no cough.

For 25 days patient was under observation and treatment, being given tonics, and bismuth mixtures for the diarrhœa. Fæces were examined some four times by same student, who reported only Ascaris ova to be present. Finally examined by Dr. COLE, who found quantities of *Fasciolopsis buskii* ova, and treatment was forthwith commenced. Fæces greyish and bulky.

April 10.—After 45 grs. thymol 11 flukes passed: average, 15·7 mm. × 10·2 mm.

Ova seemed to be of two sizes:	average of 30 large ones	= 123μ × 77μ.
Small and large lying side by side:	average of 13 smaller	= 78μ × 52μ.
Possibly mature and immature.	average of total 43	= 109μ × 69μ.

April 11.—Thirteen additional flukes: average, 20·8 mm. × 10·2 mm.

April 12.—Seven more passed, some much larger than before: average, 28·5 mm. × 11·7 mm. Of these the two largest measured 42 mm. × 1·75 mm. and 40·25 mm. × 12·25 mm.

April 21.—After two courses of β-Naphthol (B. W. & Co.), grs. 45, each divided into three doses, without the use of a purgative, three more flukes passed; average size = 19·4 mm. by 8·6 mm.

April 24.—Diarrhœa still continuing, four more passed per anum: average size = 28·4 mm. × 11·4 mm. Patient says he is immensely improved; his skin no longer dry.

April 28.—After another course of thymol, grs. 45, patient evacuated 128 flukes of the smaller type. Not measured.

April 29.—Eleven more flukes to-day.

May 5.—Another course of thymol, grs. 45, resulted in 180 more flukes being passed. They seemed much larger parasites: average, 28 mm. by 10·75 mm. (Only 21 measured out of 180.)

May 6.—No less than 71 more passed to-day, all very small, Average of ten measured, 17·7 mm. by 10·40 mm.

May 12.—Ova having been found in fæces, another course of thymol. Measurement of ovum = 150μ × 100μ.

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May 14.—After a whole day without a motion 27 more flukes passed, large and small kinds mixed. Nearly all were whitish yellow from commencing digestion in bowel.

May 21.—Ova still present in fæces. Calomel, grs. 6, at night, followed by thymol, grs. 45, on empty stomach in morning in three doses at an hour's interval, and white mixture ʒi subsequently. Twenty-six more flukes passed.

June 3.—Three successive daily examinations of fæces have shown no ova, and as patient seems a new creature, and fairly fit, he is allowed to return to College, having passed a grand total of 485 flukes whilst under treatment, six distinct courses of thymol or β-Naphthol having been given. Thymol seemed to give better results.

CASE 972. Case under Dr. G. J. EVANS, Hangchow. September, 1911.

Man. æt. 31, admitted for distension of abdomen and general discomfort. History that the distension had been gradual, and dated back some two years; that he had had some fever, but he had not been laid up by illness; during the last four months abdomen increasing in size rapidly.

On admission there is definite ascites, the abdomen is generally enlarged, spleen and liver not palpable, heart and lungs normal. Urine acid, no albumen or sugar, specific gravity 1023. Pulse 76. Not specially anæmic, does not appear "ill." Fæces are yellowish-green and well formed, and contain ova of Fasciolopsis species. No history of passing blood, and he denied diarrhœa in the past two years.. No œdema of legs and no dyspnœa. He feels discomfort after a meal, not specially in epigastrium.

He was given on an empty stomach the following:—

Oleum eucalypti ...	ʒss	Repeated after half an hour.
Chloroform ...	ʒss	
Oleum ricini ...	ʒss	

He passed the same day about 100 flukes of very varying sizes and shapes, of which the longer type (usually called *Fasciolopsis buskii*) predominated. Dr. EVANS selected three specimens of each of three types, and, except for washing them in rainwater, measured them untreated in any way.—

Type A—"Longest"	50·8	millimetres	long.
	50·8	,,	,,
	53·3	,,	,,
Type B—"Medium"	38·1	,,	,,
	44·3	,,	,,
	38·1	,,	,,
Type C—"Shortest"	25·4	,,	,,
	27·9	,,	,,
	27·9	,,	,,

In addition, Dr. EVANS measured ova from the uterus of each of these three types, and the average of ten ova in each case was as follows:—

Type A—"Longest."	Average of ten ova	156μ × 99μ.
Type B—"Medium."	,, ,, ,,	142μ × 97μ.
Type C—"Shortest."	,, ,, ,,	142μ × 102μ.

The note was made that the measurements of the ova in the fæces before treatment was practically identical with the average of the ova removed from the uterine coils.

Specimens of *Fasciola hepatica* from cattle (Shanghai slaughter-house):—

1.	Very long fluke.	Average of 14 ova from uterus	= 190μ × 105μ.
2.	Medium-sized fluke.	Average of 20 ova from uterus	= 182μ × 112μ.
3.	Short fluke, No. 1.	Average of 7 ova from uterus	= 168μ × 109μ.
4	Short fluke, No. 2	Average of 2 ova from uterus	= 167μ × 88μ.

DISCUSSION.

Colonel CLAYTON LANE: I would like to make a few remarks on this paper, and, first, as to the unity, or otherwise, of the species. GODDARD has recently done much work on the matter, and he has had a larger quantity of material, fresh and preserved, than, I think, anyone else except Dr. COLE has had; and he has concluded that between *Fasciolopsis buskii*, *F. rathouisi* and *F. goddardi* there is a regular series of gradations, so that there is only one species, namely, *F. buskii*. With regard to

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F. fulleborni, that was a rather short, broad fluke found in a Mohammedan from Calcutta, and its main characteristics were that it had no cuticular spines, that the cirrus pouch, instead of being straight, was coiled, and contained a vesicula seminalis which had no cæcum. LEIPER examined the original material, and discovered evidences of spines on the cuticle. I have, from Assam, material which fits the external characters of *F. fulleborni*, and has a coiled cirrus pouch, but the vesicula seminalis has the ordinary cæcum, and cuticular spines are present. This is another instance of a species being designated on the characters of a single individual, and that individual obviously partly decomposed. It is an observation which must be regarded as inaccurate, and the species cannot stand. My opinion is formed on reconstruction from complete serial sections.

The *Code of Zoological Nomenclature* has two contradictory articles in it. The first is that the original name given to a species shall be its valid name, and the other is in cases of patronymic specific names they shall be formed by the addition of *i*, not of *ii*. Accordingly the contradictory articles were referred to the International Commission on Zoological Nomenclature, and they ruled, in 1910, that the law of priority holds every time. The specific name, *buskii*, was the original name, given by LANKESTER in 1857, and that is the valid name. There is no hope of settling nomenclature unless one follows the Code.

With regard to the method by which these flukes produce their bad effects, there is much to be said for the belief that they do it by direct blood loss. The fluke has a big acetabulum, so big that it cannot fit within the fluke from dorsum to venter, but has to be curved posteriorly. In addition to the unusually strong musculature of the anterior part of the alimentary canal, it has also a powerful body musculature, so there is presumptive evidence that the fluke is capable of blood-sucking, and, in addition, it is blood coloured. It is difficult to distinguish it from blood-clot in the stool. GODDARD described it as ham coloured, and this evening it has been described as beef coloured. What causes the damage seems to be a direct loss of blood.

Another point is as to whether the symptoms attributed to infestation are produced by infestation. GODDARD'S figures for 1919 show that in 884 stool examinations the ova of *F. buskii* were present in 45·7 per cent., so, according to that, nearly every second person in Shao-hying is infested. Is it legitimate, under those conditions, to put down the symptoms to the infestation? Disinfestation produces extraordinarily rapid amelioration of symptoms, and one would say that this is proof that the symptoms were caused by the infestation. On the other hand, the symptoms are very much like those of ankylostomiasis, and the drug which removes fasciolopsis equally removes ankylostomes. GODDARD found ankylostomes in only 2·9 of his 884 examinations, so that there is no evidence that ankylostome infestation is the cause of the symptoms, and one is justified in attributing them to fasciolopsis infestation. It is an infestation which produces considerable damage and disability in the area in question. This fluke is, however, very widely distributed, and probably produces much more trouble than is realised. I think this paper is likely to be extremely valuable.

Dr. J. POWELL: With regard to the difference in the species, I remember, some years ago, Colonel LISTON showed, in Bombay, a specimen of what he called *Fasciolopsis fulleborni*. It occurred to me it was the same worm as I found in five cases in Assam. I took some of my specimens, and, with the aid of the text-book, we went over some of the points. We found LISTON'S cases corresponded with *F. fulleborni* and mine with *F. buskii*. I do not remember the particulars, but we were satisfied then that they tallied with the text-book descriptions. With regard to symptoms, there was ankylostomiasis in all five, and, in addition, three had round-worms. I attributed the symptoms to ankylostomiasis.

Dr. G. M. VEVERS: NAKAGAWA, the Japanese worker, recently claims to have discovered the intermediate host and development of *F. buskii*. He states that the Miracidium enters fresh-water snails of the genus Planorbis (*P. cœnosus* and *P. largillierti*), and in the liver of the snail undergoes development into a sporocyst. Within the sporo-

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cyst rediæ are formed, which in turn give rise to the infective bodies or cercariæ. The cercariæ leave the snail and encyst on grass or other vegetable matter. Infection with the adult fluke is acquired by eating the encysted cercaria on raw vegetables.

In its development *F. buskii* resembles that of *Fasciola hepatica*, the liver-fluke of sheep.

Dr. A. F. COLE: I am sorry there has not been more discussion, but I suppose it is natural, as it is not every day that we have an opportunity of seeing patients with this rare worm. Judging from the remarks of Colonel CLAYTON LANE about Dr GODDARD, whom I know well, it is not everywhere that the worm is rare. Still, I had no idea that 45 per cent. of the Shao-hying people were infected.

As regards Dr. GODDARD'S paper, which I heard of for the first time from Colonel CLAYTON LANE, it seems to point in the same direction, that is to say, there has been differentiation into species which is not quite warranted, and I suppose the same thing occurs in many other branches of zoological science. With regard to ankylostoma as a concurrent infection, I think we can exclude it in the case 729, in which 485 flukes were counted; I did not myself count every one—I sometimes left the counting to my dressers, but large flukes like this are not easily missed.

I am interested to learn there is only one species suggested in GODDARD'S paper. I was hoping Colonel CLAYTON LANE would have told you what he told me, that it was the American, WARD, to whom these specimens were sent, and it was he who described the species *F. goddardi*, which Dr. GODDARD now seems to doubt.

It is not impossible to have a multiple infection, but, on the whole, I think, the evidence points to the fact that there has not been justification for the description of several species.

With regard to the infection in India, which Dr. POWELL spoke of, I remember, in 1911, coming through India and going to see Sir LEONARD ROGERS, and, because of his vast experience, asking him what his experience was, and, as far as I remember, he had not met with any cases. Possibly my memory on the point is at fault. It must be very rare, and, therefore, I am interested to hear that in Bombay they have discovered cases.

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SOME OBSERVATIONS ON THE PATHOLOGY OF RELAPSING FEVER.

By Lieut.-Col. J. C. KENNEDY, R.A.M.C.

Relapsing fever is endemic in Mesopotamia, and was the cause of a considerable amount of sickness in the Mesopotamian Expeditionary Force, particularly amongst Indian troops and followers.

The seasonal incidence was very marked, as more than half of the total cases for the year occurred in the first quarter (1918-19), and this, of course, is accounted for by the influence of the very cold winter on the personal habits of the population. The month of the greatest incidence is March, and the lowest, October.

The disease was undoubtedly the louse-borne variety, but I am unable to say whether the causal organism should be classified as *Sp. obermeveri* or *Sp. carteri*; indeed, if such differentiation can be made, it would appear to be unnecessary for the purpose of this paper.

In this connection it must not be forgotten that a variety of the disease closely resembling African tick fever, and probably conveyed by a tick, is prevalent in Persia [1] [2]; but though it is possible that some of the cases on the Persian lines of communication may have been of this variety, there is no evidence of its occurrence in Mesopotamia.

In the beginning of 1919 a number of cases occurred in and about Bagdad, with a considerable mortality. During the period 6th October, 1918, to 26th June, 1919, the case mortality was 3·63 per cent (British, 6·49 per cent., Indian, 4·51 per cent, local, 2·53 per cent.). The predominant clinical feature of these cases was very marked cerebral symptoms (delirium, coma).

That there is a type in which cerebral symptoms predominate is not a new observation, though brought to notice by several writers [3] [4] [5] [6] recently, as it is mentioned in Vandyke Carter's monograph.[7]

The brain of one of these cases was sent to me for examination, and interested me so much that I eagerly sought the chance of obtaining more post-mortem material.

I may mention that the chief point of interest to me lay in the variety of inclusions to be found in the endothelial cells.

Shortly after, by the courtesy of the O.C. Isolation Hospital, I was able to be present at the post-mortem of one case, but unfortunately, from my point of view, it was the last fatal case that was obtainable.

Before describing the results of the examination of this post-mortem material, let me briefly consider what is the present state of the morbid anatomy and pathology of the louse-borne relapsing fever. Text-book descriptions are meagre, and the literature on the subject is not very enlightening, while some of the best work was carried out some time ago, when histological methods were crude compared with the present day. But no doubt it is the scarcity of suitable material that has handicapped research in this line.

In a very comprehensive review of the literature on spirillar fevers up to 1908, Mackie[8] says, with reference to the European variety:—

"There are no characteristic changes in the organs of persons who have died of

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relapsing fever; such morbid appearances as are met with are those generally associated with severe septicæmia. Thus, there is marked fatty degeneration of the heart muscle, whilst the spleen and liver are enlarged and show necrotic patches, which are due either to infarction or to local concentration of spirillar toxins. Hæmorrhages are not infrequent, especially in the kidneys.

"Some degree of bronchitis is constant. The bone marrow shows necrotic patches, which are apparently due to infarction."

Again, referring to Asian relapsing fever, he says:

"The very careful description of post-mortem examinations in VANDYKE CARTER'S monograph serves only to leave the impression that there are no lesions characteristic of the disease. If the tissues are stained by those methods usually chosen for revealing bacteria or protozoa, the spirilla are demonstrated with difficulty. They are seen to occupy the blood vessels and the tissues in the neighbourhood of hæmorrhages. From this I believed that the spirillum was a true blood parasite and lived only in the circulating blood. More recently, by using Levaditi's silver impregnation method, this view has been modified. The spirilla were seen to be scattered throughout the liver, and not confined to the blood-vessels, though it is true no special relation to the tissue cells could be demonstrated."

Since 1908 the more important observations have been made in relation to fatal cases of the meningeal type (*e.g.*, BABES), but I have not come across any very detailed account of the histology, though BABES notes atrophic cirrhosis of the liver.

The case which provided the material for this paper was that of an Arab cooly who died in the Isolation Hospital, Bagdad, on the 15th May, 1919, and the following are the clinical notes extracted from the hospital register.

Admitted 15th May, 1919, from I.G.H., with a note of illness lasting four days, enlarged spleen and spirilla in blood. Aged, poorly nourished, debilitated, expression dull and lethargic. Semi-conscious, drooping of eyelids, marked jaundice. Sub-conjunctival hæmorrhage in right eye and injection of left. Tongue coated white and dry. Rash petechial, purple character on abdomen, chest, arms and thighs. No abdominal distension. Abdominal reflex present. Marked retraction of abdomen. Knee jerks absent. Heart beats soft, distant but regular. Pulse good volume, strong and regular. Lungs, left base dull, moist crepitations, V.F. and V.R. increased. At 9.30 p.m. patient collapsed suddenly after vomiting up some milk. The heart did not respond to treatment and he died at 10.20 p.m. on the day of his admission.

In view of the subsequent findings, it is rather important to ascertain the duration of the illness, but this is not possible. It can be stated, however, that death occurred after at least five days' illness.

The autopsy was performed next morning, twelve hours after death, and the following brief notes were made at the time:—

On removing the skull cap the meningeal vessels were found to be engorged with blood, and the pacchionian bodies were more prominent and more adherent than normal. The surface of the brain was very congested, and the subarachnoid spaces in the sulci were filled with extravasated blood. This was most marked on the vertex, on the occipital lobe and on the mesial aspect of the hemispheres. The ventricles contained blood-stained fluid, and the choroid plexus was congested. The vessels of the internal capsule were engorged. The spleen was very large and dark in colour, but not particularly friable. The liver was very large and fatty in appearance. The kidneys were enlarged.

The following specimens were removed for detailed examination, heart's blood and portions of brain, spleen, liver, kidney and bone marrow.

EXAMINATION FOR SPIROCHÆTES.

No spirochætes could be found in the heart's blood though they were reported present in the blood on the morning of death. Indeed, microscopic examination of smears and sections failed to reveal them, except in the case of the brain, when they were found in

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smears of the surface of the brain and in sections lying in the superficial layer of the cortex.

Examination of the Brain.

The appearances may be summarised thus :—

1. Great engorgement of the meningeal vessels, and, to a less extent, of the vessels passing into the cortex.
2. Extravasation of red cells into the perivascular spaces of the pia-arachnoid with a few round cells and polynuclears.
3. Hæmorrhages on the surface of the cortex immediately under the pia mater.
4. Small areas of round cell infiltration in the same situation as in 3.
5. Proliferation of large mononuclear (endothelial) cells, particularly in the perivascular spaces round the meningeal vessels.
6. A great deal of pigment.
7. A few spirochætes, as detailed above.

The pigment was of two kinds, (*a*) a golden brown pigment in needles or rhomboids, and (*b*) an amorphous brown pigment. Both kinds were found lying free or phagocyted by large endothelial cells. A large amount of the crystalline form was lying free in the vessels or attached to the endothelium, and in spaces containing extravasated blood. The amorphous form was for the most part inside large endothelial cells, or collected in large black masses in the perivascular connective tissue.

The extraordinary proliferation of the endothelial cells was the most striking histological feature. This was particularly marked in sections of the cerebellum, and in smears from the surface of the brain these cells showed up in large numbers. They were actively phagocytic, containing pigment, red cells and even polynuclears ; some were absolutely gorged with red cells. Some were full of vacuoles, and the protoplasm stained irregularly an olive or greenish-blue tint with Romanowsky. A considerable number contained spirochætes.

As mentioned above, spirochætes were found only in the brain. In sections stained by Levaditi's methods, it was only in the superficial layer of the cortex that they could be found, and then in very scanty numbers. In smears from the surface of the brain a few were lying free, but more often were inside endothelial cells. Most of them stained faintly and irregularly (*cf.* stage of disease). An occasional one presented an appearance suggesting Leishman's "buds," but a large number, and those chiefly intracellular, had a granule or small spore-like body attached, which was usually terminal.

Polynuclear leucocytes attracted attention, for the reason that many of them contained small round or oval bodies, of very uniform size, which could not be stained by any of the methods employed—Romanowsky, Heidenhain or silver methods. They were probably digestive vacuoles, but they gave one a different impression, and the appearance of a cell containing a number of these uniformly sized bodies (or vacuoles) was unusual.

Examination of the Liver.

The histological changes in the liver were striking, and may be enumerated as follows :—

1. Great destruction of the liver cells, the periphery of the lobule being the least affected.
2. Infiltration of round cells into Glisson's capsule.
3. Engorgement of the intralobular capillaries.
4. Proliferation of large endothelial cells.
5. Deposition of a considerable amount of pigment.
6. The presence of numerous round or oval bodies, the nature of which I am unable to determine.

The destruction of liver cells is the most marked feature, and is most evident in the

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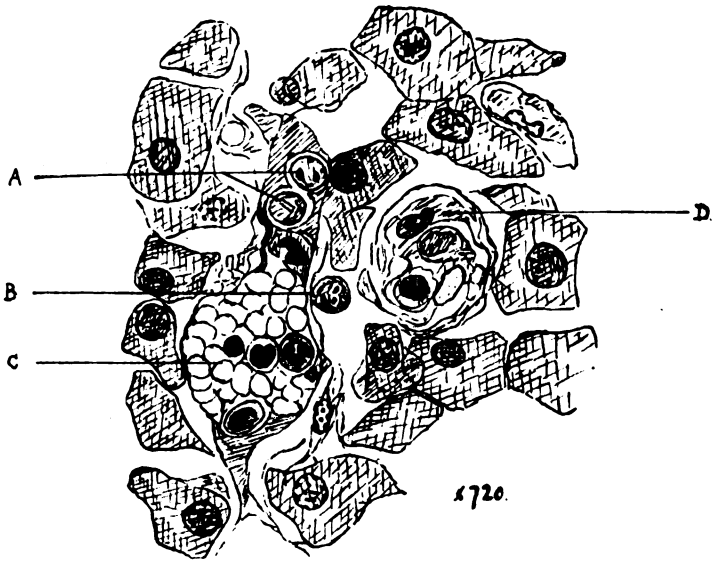
centre of the lobule, the columns of liver cells being replaced or displaced by distended capillaries or blood spaces, in which large endothelial cells or Kupffer cells are prominent. This presumably is the condition noted by earlier workers (CARTER in India and PONFICK [9] in Europe). and described as a necrosis due to infarction or to the action of toxins.

The vacuolated appearance of the degenerating cells suggests fatty degeneration but I was unable to demonstrate it with osmic acid, On close examination one was led to wonder how much of the degeneration was primary and how much secondary to an endothelial proliferative reaction as the result of which the columns of liver cells are invaded and broken up. At the same time the gland cells show vacuolations, pigment inclusions, inclusions of other bodies of undetermined nature, fragmentation and nuclear degeneration.

The next thing that strikes one is the capillary engorgement and the prominence of large endothelial cells of Kupffer type. Under the low power, cross sections of these dilated capillaries containing Kupffer cells are prominent, and remind one of "cell nests." With suitable staining the Kupffer cells are seen to contain a variety of inclusions, pigment, leucocytes, red blood cells and inclusions of undetermined nature.



CAMERA LUCIDA DRAWING OF A SECTION OF THE LIVER FROM A CASE OF RELAPSING FEVER, DEMONSTRATING—

A—Cell inclusions of undetermined nature (?altered leucocytes in Kupffer's cell).

B—Free "body" of undetermined nature.

C—Distended capillary blood-vessel.

D—Proliferation of endothelium—Kupffer's cell with inclusions.

The pigmentary deposit in the liver is considerable; it is dark brown to black, and arranged in granule clumps, or in circles or spheres with a clear centre, giving a "signet ring" appearance. It is found in the liver cells but for the most part lying free in spaces amongst cellular debris or phagocyted by endothelial cells.

The epithelium of the bile ducts was unaffected, though there was some round celled infiltration in the capsule.

Being of the opinion that the relapses of spirillar fever can best be explained by a cycle of development, and that the clue to this cycle is probably the granule, I studied this liver condition carefully, endeavouring to find some indication of a granule phase.

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This idea occupied my mind to such an extent, that it was some time before I realised that certain bodies which I had passed over as altered leucocytes were in fact something quite distinctive, and unlike anything of a histological nature that I was acquainted with. A further examination revealed the fact that these bodies could be found inside the liver cells, chiefly towards the outer parts of the lobule where the liver cells had not undergone such extensive degeneration. In the more degenerated portions at the centre of the lobules these bodies were quite numerous. They are generally to be found lying free, or apparently so, in relation to degenerating gland cells, or in capillaries or inside Kupffer's cells. Their reaction to ordinary stains is feeble, but their structure can be fairly well picked out by Heidenhain's or Weigert's iron hæmatoxylin, or deep staining with Leishman's stain.

I was unable to determine the nature of these bodies and came to the conclusion that they could not be leucocytes or degenerating nuclei, and thereupon, perhaps somewhat hastily, sent a note to the *R.A.M.C. Journal*[10], suggesting that they were protozoal bodies, possibly having some relation to the spirochæte, and waited expectantly for more post-mortem material.

However, I have not been able up to the present to extend this research, and recently returned to a careful reconsideration of the original material, with the result that my faith in the accuracy of my former deduction has been shaken. In this state of mind I showed specimens to Dr. WENYON and Dr. STEVENSON, whose criticisms, though kindly, did not help to dispel my doubts.

If these bodies are not of a specific nature, then they are probably a phase of degeneration of polynuclears, but there is to me something unique in the characters of the nuclei when well stained that I find difficult to attribute to degeneration.

It is obvious, therefore, that no useful purpose will be served by doing more at present than note the presence of these bodies, and I content myself with showing the specimens under the microscopes in the hope of obtaining further enlightment from the members.

With regard to the spleen, the kidney and the bone marrow, I have nothing further to add to what is already known and described.

In the spleen the amount of pigment deposited is very great, the "signet ring" form being prominent; the presence of necrotic areas and of large phagocytic endothelial cells is noted.

I am afraid that these observations are not of a startling character, but they are perhaps sufficiently interesting as indicating two points in the pathology of relapsing fever which will repay further study, viz.:—

1. The reaction of the endothelial cells.
2. The histological changes in the liver.

The endothelial reaction is a feature of some protozoal infections—notably leishmaniasis—and we are tempted to cite it as a point in favour of the protozoal nature of spirochætes.

The changes in the liver may be simply the result of toxic action combined with a reaction of the endothelial elements, but in the presence of such marked histological changes one is led to hope for the key to the life history of the parasite.

Keeping this point in view, and in conclusion, let me quote VANDYKE CARTER'S summary of his observations on the condition of the liver in 74 post-mortems:—

"The minute structural changes which I have seen consist chiefly of cloudy swelling and pigmentary or fatty transformation of the gland cells. The extremely localised degeneration sometimes evident may be peculiar to this infection, and it would indicate stasis of circulation in the finer branches of the portal vein.

"As to the cause of morbid alterations, there is, earliest, enlargement of the liver with uniform or disseminated pallor, and friable consistence, all of which quickly supervene on the pyrexia, and are most pronounced in the first attack. At this time there is a general turgescence of the gland cells, probably general, and the interlobular connective tissue is only seldom implicated; fatty transformation of the cell substance is yet scarce.

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"With cessation of pyrexia the turgid condition subsides, or it passes into a condition of fatty metamorphosis, which I have found to be commonly present in casualties at post-invasion periods, whether or not accompanied by cloudy swelling of the cells.

"Recurrence of pyrexia entails repetition of enlargement (though to less amount) and gland cell alterations. Finally, the organ tends to resume its normal dimensions, but traces of cell lesion may long persist."

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DISCUSSION.

The PRESIDENT: Colonel KENNEDY is to be congratulated on the very careful and complete record he has made of the pathological changes found in this one post-mortem examination, and I hope he will have opportunities of pursuing his investigations further, after the Fellows of this Society have had the opportunity of commenting on his work.

Dr. C. M. WENYON: As Colonel KENNEDY was kind enough to show me his preparations the other day—which I examined in consultation with Dr. STEVENSON—perhaps I may say a word about the bodies he had been directing attention to. Apparently the case of relapsing fever he has been talking about was a very severe one, so that the changes must not be regarded as the ordinary ones which occur in the disease.

With regard to the particular bodies which have been found in the endothelial cells, I cannot give a suggestion as to their nature. In the endothelial cells there occur polynuclear leucocytes, red-blood corpuscles and other things. We do not always recognise the extraordinary phagocytic power of the large endothelial cells in some acute infections. They are not limited to the blood-vessels. They break loose from the walls of the vessels and migrate through them, and wander about the extra-vascular tissues. They seem to be ravenous cells, ingesting anything which comes their way, whether polynuclear leucocytes, red-blood corpuscles, or bacteria. They have powerful digestive properties, and digest these cells. Some years ago I injected a mouse intraperitoneally with material containing leishmania, and the mouse developed an enormous peritoneal exudate. I withdrew some of the fluid from its abdominal cavity, and found a large number of these large endothelial cells, and they contained an extraordinary assemblage of structures in their cytoplasm. Some were lymphocytes, others were polynuclear cells, others were red cells, and some were structures of unexplained origin. Every kind of digestive process seemed to be going on in the vacuoles. So it is not strange we should come across bodies the nature of which we cannot explain.

What I think may give rise to structures like these within the cells is this: A large endothelial cell is a very large structure; it may be twenty microns in length and fifteen microns in thickness. In sectioning tissue, one may get slices of these big cells and of the bodies which are in them. Perhaps a polynuclear cell in the endothelial cell has the top of it cut off; its nucleus may or may not be included, or only a part of it. It would be

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difficult to recognise unless one could discover the exact spot from which it came in an adjoining section of the series. Points like this can only be worked out by careful study of serial sections; taking isolated sections may give only small fragments of the included bodies, and I offer this as an explanation of the bodies Colonel KENNEDY is showing us to-night.

Dr. J. POWELL: In the years 1900-3 we had a severe epidemic of relapsing fever in Bombay, and during that time I examined post-mortem some two hundred cases. The great majority of these were people found dead in the streets and brought in by the police. I can corroborate VANDYKE CARTER'S statement that the characteristic of the post-mortem examination is that there is no one definite sign to indicate that the condition is relapsing fever; the appearances were those of an acute septicæmia. The most striking points were:—

(1) The absence or early disappearance from the blood of the spirilla.
(2) The *enlargement* of the spleen and liver, which was constant.
(3) The *softness* and friability of both these organs.
(4) The frequence of fatty degeneration of heart, spleen, liver and kidneys.
(5) The fluidity of the blood. Any clots found in the right heart were, as a rule, small, broken up and dark, in contrast with those found in pneumonia, influenza, and, to a less extent, in plague.
(6) The frequence of small hæmorrhages, and
(7) of jaundice. Even when no jaundice was observed in skin or conjunctivæ, the serum of the pericardium, pleura and brain was often yellow and bile-stained.

In some cases there was evidence of bronchitis. The proportion of cases in which I found pneumonia was small, but my observations at this point are of little value, as in the case of dead bodies with no history, with no spirilla to be found, with definite pneumonic signs, I, perhaps excusably, recorded the cause of death as pneumonia.

At that time, too, there was plague in the city. When people were brought in dead, as a rule, the cause was either plague or relapsing fever. If no plague bacilli were found, by a process of exclusion we said it was relapsing fever. Even when the post-mortem was done in the afternoon of the day the body was brought in, we could not find the spirilla; they seemed to disappear very soon after death. To find out at what time they disappeared, I examined twenty-five cases dying in hospital under observation with spirilla in the blood shortly before death, and the longest period after death that I could find the spirilla was seven hours, and then in only one case. It was unusual to find any two hours after death, though before death the spirilla had been present in large numbers. The fluidity of the blood in these cases was striking. I know of no death from asphyxia or plague in which the blood is so fluid as in relapsing fever.

It seemed to me that in relapsing fever the spleen was much softer than we find it in malaria or in plague. The liver was always enlarged and friable. Another thing which made it difficult to distinguish relapsing fever from plague was the frequency of hæmorrhages, especially under the pleura, pericardium and other serous membranes. As regards fatty degeneration, I think this depends on the period at which the patient died, It is common to see fatty degeneration of both liver and heart, but it is not so striking if the victim has died in the first attack, when we find only cloudy swellings, In a case in which a man died suddenly of syncope after his third attack, the condition of the body was strikingly like that of a case of delayed phosphorus poisoning. There was extreme fatty degeneration of the heart, spleen, kidneys and liver; they were light yellow, and there were dotted hæmorrhages over the pericardium, just as if some one had dipped his finger in blood and had flicked it over these regions. I can best describe the condition like an Irishman, as typical of acute yellow atrophy of the liver except that the organs were hypertrophied. I regret that my post-mortems, though so large in quantity, were so poor in quality; but my duty was medico-legal—to ascertain what the patients had died of and dispose of the bodies.

With regard to the pigment which has been found in these cases, I suggest that much of it was due to previous malaria infection, and I ask the author's view as to that. The

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delirium in this disease is often of the type of a *wanderlust* associated with terrors, under the influence of which the patient gets up and leaves home, wanders some distance away, curls up, and dies on the roadside; most of them were very poor, and without nursing assistance.

Dr. W. H. WILLCOX: I am particularly interested in Colonel KENNEDY's paper, because I was in Mesopotamia for three years before Colonel KENNEDY arrived, namely, from 1916 to 1919, and I saw a great number of cases of relapsing fever there. Captain S. W. PAGE, of the Isolation Hospital, Bagdad—who, I believe, made the post-mortem examination on this case—said, in a letter to me from Mesopotamia, that since I had left there was a remarkable change in the type of case they were having—that a number of very acute cerebral cases had occurred having a rapidly fatal result. I referred to these cases in a paper which I read before the Royal Society of Medicine in January, 1920. In 1918, the mortality from relapsing fever was very much less than the figures which Colonel KENNEDY has quoted, so that the type had much changed in virulence. The figures were 8 per cent. in Indians, 1 per cent. in Arabs, and no Britisher died there in 1918 of that disease. In 1916–17 as well as 1918 there was relapsing fever, and nearly every case was traced to infection from Turks or Arabs. I saw a number of cases of relapsing fever in Turks in 1917, when so many of them were captured.

Colonel KENNEDY referred to relapsing fever in Northern Persia. I spent six months there in 1918, along the line of communication from Bagdad to Enseli, and the relapsing fever one met with there was the same in type as occurred in Mesopotamia, and could be definitely attributed to lice infection.

With regard to tick fever, I did not get quite to Mianeh, only going as far as Linjan, which is within fifty miles of it. I heard, however, much of these cases from medical officers who had seen them. It appeared that at Mianeh a large tick is prevalent, which has a great liking for strangers, who soon fall victims to its bites. It seems to spare the Persians Mianeh fever, caused by these bites, appears to be very similar to relapsing fever, and to resemble the tick fever occurring in Palestine, which was described by Dr. H. D. NICHOLSON, R.A.M.C., in 1919.

Colonel S. P. JAMES: I would like to associate myself with Dr. WILLCOX, with whom I was in Mesopotomia at the time he refers to, in congratulating Colonel KENNEDY on his paper. It is one which lends itself very well to discussion. Personally I would like to hear more discussion about the diagnosis of the fatal illness from which Colonel KENNEDY's patient died; especially one would like to hear the grounds on which it is thought that relapsing fever was the only cause of death. As malaria was also very prevalent in Mesopotamia—in the early years, at any rate, and later in Persia also—one would have thought Colonel KENNEDY would have told us whether this particular man had previously had attacks of malaria. On that point, I think, a good deal of interesting discussion might rest.

Surgeon-Rear-Admiral P. W. BASSETT-SMITH: I regret I cannot throw light on the question which has been raised; I have not had any personal experience of relapsing fever. I think that in blackwater fever one meets with the same kind of inclusions, which were first referred to by LEISHMAN. In sections and smears of organs from animals which have died of acute trypanosome infections something similar is seen too. Thus it seems possible that many poisons may, under certain conditions, give rise to this peculiar character of cell. Such a cell is not necessarily a diagnostic sign of relapsing fever, but rather of an intense toxic infection. So also are the cortical hæmorrhages, and the hæmorrhages under the pia mater and meninges found in other conditions, for instance, in anthrax.

Dr. G. C. LOW: I would like to ask Colonel KENNEDY whether he has examined other cases of relapsing fever, and, if so, in how many was this endothelial proliferation present? The inclusions in the cells are not similar to those one finds in blackwater fever

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cases. I agree with Dr. POWELL and Colonel JAMES that some of the pigment present in the sections is of a malarial nature—at least it is difficult to say it is not. It may be that this man was a malarial individual who died of relapsing fever, and that is the reason of so many endothelial cells being present. The way to prove this point would be to examine cases of relapsing fever in non-malarial districts, and histologically compare the two sets. I agree with Dr. WENYON that if there are so many endothelial cells with all sorts of inclusions, and if you look at some of these in cross-section, you are certain to see all sorts of appearances which it will be difficult to interpret. I think these bodies are leucocytic in nature, degenerative probably, or seen in cross-section.

Lieut.-Colonel J. C. KENNEDY: Answering several members in regard to malarial infection, I agree it is very likely a large amount of the pigment is due to malaria, but there was no indication of any malarial parasites being present, though I looked carefully in the spleen and the brain and in the smears. I think the majority of it was due to the infection by the parasite.

In answer to Dr. LOW, I thought I had made it clear that I have not had experience of post-mortems in relapsing fever beyond what I have related to-night. It proved so interesting to me, and I could find little in literature to help me. I, therefore, thought discussion of the matter might be of interest to the Society.

I was very interested to hear Dr. POWELL's experiences in Bombay; they bear out exactly what I gathered from Dr. CARTER's monograph. CARTER also brings out the point that the fatty degeneration of the liver is most marked towards the second or third relapse.

With regard to Dr. WILLCOX's remarks, there was undoubtedly a change in the character of the infection in Mesopotamia, and I am aware that the mortality amongst the British was negligible until this outbreak occurred. With regard to Mianeh tick fever, a very interesting account of that is given by Major HAROLD in the *R.A.M.C. Journal*, and he points out the special predilection of these ticks for foreigners.

Surgeon-Rear-Admiral BASSETT-SMITH referred to blackwater fever, but I think the inclusions in that differ from those in relapsing fever. And the hæmorrhages are not, as he says, specific; they are merely evidences of the great toxicity of the infection. What specially interested me was this great endothelial reaction, and I think it will be worth while to seize every opportunity to study that special point in connection with this disease.

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OBSERVATIONS ON FILARIA IN CHRYSOPS FROM WEST AFRICA.

By A. CONNAL, M.D., D.P.H., *West Africa Medical Service.*

This communication is of interest, in view of the suggestion made by Sir PATRICK MANSON, many years ago, that *Chrysops dimidiata* v. d. WULP might prove to be the carrier of *Loa loa.*

LEIPER, in Nigeria, and KLEINE, in the Cameroons, have both given support to that theory.

The observations herein recorded were made during 1917, at the Medical Research Institute, Lagos. They are of a preliminary nature, and they show that Chrysops harbour filaria. No feeding experiments were done and no sections were made. The work consisted entirely of dissections, which were carried out by my wife. The material examined amounted to 2255 specimens of Chrysops. Two species were represented, *silacea* (AUST.) and *dimidiata.* They were collected within a mile or two of Sapele. Sapele is a trading centre, on the creeks, about two days' journey by launch from Lagos eastwards. The district has the reputation of being heavily infested by the so-called "mangrove flies." Incidentally this popular term includes several different genera of blood-sucking diptera. The inhabitants, both European and native, show a considerable percentage infected with *Loa loa*, as evidenced by the presence of embryos in the peripheral blood; many of the Europeans also contract Calabar swellings.

Mr. METHVEN, in charge of a rubber plantation on the outskirts of Sapele, very kindly arranged for his native staff to collect the flies. He himself undertook the preserving and forwarding. The preservatives used were formalin 10 per cent., glycerine or spirit. All the dissections were done with the aid of the binocular dissecting microscope.

The actual figures were published in the Annual Report of the Medical Research Institute, Lagos, for 1917. They are summarised as under :—

Month.	Silacea.	Infected.	Dimidiata.	Infected.
March ...	3	0	7	0
April ...	87	2	44	3
May ...	287	1	30	0
June ...	679	2	25	1
July ...	567	6	41	0
August ...	189	2	19	0
September..	117	1	26	0
October ...	77	2	57	2
Eight months	2006	16	249	6

Percentage infected, silacea = 0·8
" " dimidiata = 2·4
" " 2255 flies = 0·9

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May, June and July are the wettest months. The largest number of flies was collected during this period.

The positive flies were practically always heavily infected. As a rule the filariæ were present in hundreds; 427 was the actual count in one insect. Had sections been made and suitably stained, possibly a higher percentage of infection would have been found. The worms were situated for the most part in the thoracic muscles. Two flies were obtained which showed filariæ amongst the mouth parts. (Microphotographs of the mouth parts of these two flies were exhibited.)

In Sapele, then, there exist (1) a population who harbour adult *Loa loa* in their tissues and loa embryos in their blood; (2) a large number of Chrysops, a certain percentage of which are infected with filaria.

Discussion.

The President: Dr. Connal is to be congratulated on having a wife who has been so helpful to him in carrying out these important investigations. I know how thorough her work was when she was at the London School of Tropical Medicine, then located at the Docks, and when she was assisting Professor Alcock there. She has done excellent work, not only in this particular investigation, but in many others, and what she does is always very reliable.

I have nothing to say on this subject, further than I hope that Dr. Connal will continue his researches into the breeding-places of the flies, and the connection they bear to the prevalence of *Filaria loa*.

Dr. G. C. Low: I wish to thank Dr. Connal for bringing forward these very interesting observations. As all who have done work on intermediate hosts in filariasis know, the difficulties are very great. To be absolutely certain of one's results, one must rear all material from the larva, so that one knows that one is starting with a clean, uninfected stock. When dealing with rare insects this is often quite impossible, but, even so, one can catch such samples wild and feed them on the filarial case, and if one finds nothing it means that that individual insect is not a host. If, on the other hand, one finds developmental forms of filaria present eventually, then one cannot say definitely that the insect has not acquired an infection naturally from some animal or bird, and the result must be dubious.

This is the position Dr. Connal is in with the Chrysops he tells us of. Nevertheless, it looks as if Chrysops was the proper intermediate host, because of the geographical distribution corresponding so closely. Dr. Connal has told me privately that where there are no Chrysops there are no *Filaria loa* infections, and, conversely, where there is much loa infection—Calabar swellings, history of worms in eye, etc.—there such flies swarm. Dr. Connal has told us that the developmental forms occur in the muscles of the fly, a very interesting observation, and one which brings the development into harmony with that of *F. bancrofti* in the mosquito, *e.g.*, a muscle developmental form and not a Malpighian developmental one, like *Dirofilaria immitis* of the dog. Again, his forms escaping from the proboscis measure one-sixteenth of an inch, a similar measurement to those of *F. bancrofti* escaping from the proboscis of the mosquito. I hope Dr. Connal will be able to go on with this work, and I would suggest to him that he try rearing Chrysops in captivity to get a clean stock, and then to feed them on cases of loa infection with embryos in the blood. He can then dissect the flies daily to watch the development, and can cut sections of them to see exactly where the development takes place. By such means it will be possible to finally decide the position *C. silacea* and *C. dimidiata* play as intermediate hosts for *F. loa*, a suggestion first brought forward by Sir Patrick Manson on geographical grounds.

We all, I am sure, will look forward to the results of these enquiries with the greatest interest, and hope Dr. and Mrs. Connal will be successful in their quest.

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LIST OF FELLOWS.

The following were duly elected Fellows of the Society:—

EUSTACE W. FERGUSON, M.B., CH.M. (Sydney), Sydney.

Major WILLIAM A. JUSTICE, I.M.S. (ret.), M.B., C.M. (Cantab.), London.

Lieut.-Col. HENRY KIRKPATRICK, I.M.S., M.B., London.

EDWARD ATHOL C. LANGTON, M.R.C.S., L.R.C.P., Brighton.

PHILIP ALAN MAPLESTONE, M.B., B.S. (Melb.), Townsville Old.

NEILS NEDERGAARD, M.D. (Nebr.), Oriente, Cuba.

ROBERT NEWSTEAD, M.Sc., F.R.S., Liverpool.

Surg.-Com. SIDNEY ROACH, M.R.C.S. (Eng.), Brading, Isle of Wight.

SADAO YOSHIDA, D.Sc., Osaka, Japan.

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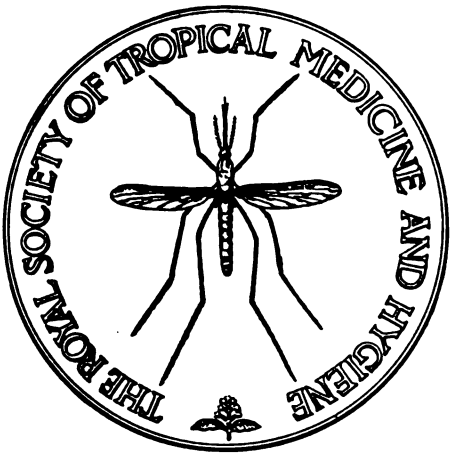

ZONAE TORRIDAE TUTAMEN

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TRANSACTIONS

OF THE

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

MARCH 18TH AND MAY 20TH, 1921.

VOLUME XV. NOS. 1 AND 2.

Proceedings of a Laboratory Meeting of the Society held, by the kind invitation of the Commandant, at the Royal Army Medical College, Grosvenor Road, S.W. 1, on Friday, March 18th, 1921, at 8.15 p.m.

LABORATORY MEETING.

Lieut.-Colonel S. P. LELEAN gave a demonstration of exhibits from the Museum of the Hygiene Department, R.A.M. College, showing methods suitable for tropical hygiene as regards urinals, latrines, incinerators, destruction of insects, trapping of mosquito larvæ, bathing, water cooling and cold storage of food, and disinfection and disinfestation.

Lieut.-Colonel J. C. KENNEDY showed a series of sections of oriental sore, illustrating its histological characters :—

(1) The formation of a tumour the cells of which are heavily infected with *Leishmania tropica.*

(2) The tumour cells in the centre of the growth have the appearance of fibroblasts, but are probably of endothelial origin.

(3) Portions of the tumour show giant cell formation.

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(4) The tumour is surrounded by and in parts infiltrated with small round cells and plasma cells.

(5) The tumour extending along the routes of lymph vessels.

(6) Hæmorrhage into the apex of the tumour, with atrophy and gradual erosion of the epidermis.

Lieut.-Colonel H. MARRIAN PERRY gave a demonstration of a helminthic infection in relation to bacterial invasion of the tissues. He remarked that attention was first directed by WEINBERG to the occurrence of bacillary invasion of the tissues in infestations of monkeys with *Œsophagostomum apiostomum.*

The present demonstration is concerned with somewhat similar pathological lesions associated with the presence of another helminthic parasite, *Galoncus perniciosus.*

The animal from which the material is derived was a leopard, *Felis nebulosa*, which died recently in the Zoological Gardens with symptoms of a hæmorrhagic enteritis simulating dysentery. Subsequent examination, however, determined the fact that the condition resulted from infestation with a helminth, *G. perniciosus*, and that death was occasioned by a septicæmia following bacterial invasion of the tissues in the neighbourhood of the parasites.

G. perniciosus is a small nematode worm, which has been encountered on several occasions in the intestine of many of the Felidæ. The worms are found encysted in small nodules in the submucous coat of the large intestine; some of these nodules are closed, whilst others present a small opening into the lumen of the bowel. In each nodule one or several worms are found lying in a mass of extravasated blood and inflamed tissue.

The pathological lesions are analogous, as noted above, to those observed in infections of monkeys with *Œ. apiostomum*, and the bacterial invasion of the tissues occurs in a very similar manner.

The contents of the submucous cysts become invaded by organisms which may cause a generalised peritonitis by rupture of a cyst into the peritoneal cavity, or a septicæmia by invasion of the blood-stream.

In the case at present under consideration, a bacterial invasion of the blood-stream was the result of the helminth infection.

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Bacillus coli and Streptococci of the fæcal type were recovered from the heart-blood, spleen and mesenteric glands of the animal.

The following specimens were demonstrated :—

(1) Ileocæcal junction showing nodules caused by the presence of the helminths.

(2) Thick section of submucous nodule showing three helminths lying in a mass of extravasated blood.

(3) Section of nodule demonstrating spread of bacillary invasion from neighbourhood of helminth.

(4) Section of submucous tissue of cæcum, with masses of hæmatoidin crystals surrounded by a zone of bacterial infection.

Major F. R. COPPINGER demonstrated a method by which he and Capt. R. C. ROBERTSON had prepared a detoxicated vaccine for *B. dysenteriæ*, SHIGA :—

The various stages of preparation and results obtained with rabbits were shown.

The vaccine was prepared by treating the bacilli with strong ammonia and then neutralising with sulphuric acid. The ammonium sulphate formed helped to bring down the altered bacilli in a heavy flocculent precipitate, which, when washed and suspended in saline, formed the vaccine.

It was noted that the bacilli were not broken up or dissolved by this process, as in the case of THOMSON'S detoxicated vaccines; the only change noted microscopically being a swelling and distortion of their bodies.

Charts, demonstrating the effect of injecting various doses of this vaccine into rabbits, were shown, from which it appeared that the toxicity of the bacilli was considerably reduced, while their power of producing immunity was not appreciably altered. A prolonged series of human experiments is necessary before a definite conclusion as to the value of this vaccine can be obtained.

Professor J. W. W. STEPHENS sent the following exhibits :—

(1) Photographs of claw hand of a patient suspected to be a case of leprosy. No bacilli had, however, been demonstrated in a portion of the thickened internal cutaneous nerve removed at operation.

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(2) Four cases of Ainhum toe:—

(*a*) A very early condition.

(*b*) Groove well marked, with outward rotation of the toe and atrophy of the nail.

(*c*) Spontaneous amputation, a condition which was bilateral.

(*d*) Involvement of the fourth and fifth toes in a Kroo-man. Here, again, the condition was bilateral.

Professor WARRINGTON YORKE sent specimens illustrating the development *in vitro* of *Strongyloides stercoralis.*

Dr. PHILIP MANSON-BAHR showed a series of sections of kidneys and spleens illustrating the pathology of a septicæmia, or pyæmia, due to *Bacillus coli.* This very grave disease, attended by a high mortality rate, occurred amongst Turkish prisoners in Egypt during 1916. Seventeen cases had been studied and nine autopsies performed. The clinical and pathological studies based upon the material have already been published in the *Lancet*, in 1918, vol. 2.

At autopsy the kidneys were found to be much enlarged, weighing as much as 14 oz. each; the cortex was studded with numerous small yellow abscesses originating from the Malpighian corpuscles. Secondary abscesses were also found in the spleen and liver. In microscopic sections it was possible to trace the path of the colon bacilli with great nicety. Large aggregations of these organisms could be demonstrated in the intertubular veins, whence they penetrated through the epithelium of the collecting tubules, gaining access to their lumen, and subsequently appearing in the urine.

From a clinical point of view these cases presented a very puzzling appearance. At first sight, some were apt to be mistaken for enteric fever; others suffered from severe recurrent rigors, which much resembled those of malaria in their duration and general course. It was possible to isolate the bacillus from the blood-stream only during the rigors but not during the quiescent periods.

In some cases there were symptoms referable to the urinary system, such as loin pains and frequency of micturition; in others these were completely absent. The urine itself presented a normal appearance, and neither albumen nor abnormal cells were present. The appearance of the bacillus in the urine was also intermittent, so that in order to

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diagnose this condition with certainty the urine had to be examined on several different occasions.

The classification of these bacilli was a difficult matter. It was possible to subdivide them on their sugar reactions into several sub-varieties, but there appeared to be nothing attained in doing so, for their pathological effects upon man and laboratory animals in each case were the same.

The entry of the bacillus into the blood-stream would appear to take place through a breach of surface in the intestinal canal; in these prisoners infection appeared to take place through old dysenteric ulcerations of the large gut.

In addition to *B. coli*, septicæmias and pyæmias due to *B. pyocyaneus* were encountered, and microscopic sections were exhibited of one case from which this organism was isolated in pure culture from renal and prostatic abscesses.

The debilitated condition of these subjects predisposed to secondary infections of all kinds, and a slide was exhibited of pneumococci ingested by a polymorphonuclear leucocyte in the peripheral blood from a case of pneumococcal septicæmia. This case was diagnosed while the film was being examined for malarial parasites.

Miss E. M. BAXTER and Dr. A. J. EAGLETON gave a demonstration of the degree of sterilisation produced by hot oil. It was pointed out that the use of oil for sterilisation of syringes is at present very general. Sir ALMROTH WRIGHT described a technique for the laboratory and the field, and states that "a temperature of 125° C. to 140° C. will sterilise instantaneously everything with which it comes in contact."[1] Professors DREYER and AINLEY WALKER concluded, from a series of experiments, that a temperature of from 150° C. to 170° C. for not less than half an hour is necessary.[2] More recently, Dr. WATERHOUSE[3] has claimed that the method is valuable.

The importance of the subject is very great, especially to workers in outlying districts who need a simple and efficient method of sterilising syringes. The stress laid on the dangers of quinine injection in the tropics is familiar to everyone.

Four types of experiments have been carried out :—

(1) Subjection of sporing *B. subtilis* to liquid paraffin and olive oil at different temperatures.

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(2) Infection of syringes (all glass) with *B. subtilis* cultures. Olive oil and linseed oil and liquid paraffin, at different temperatures, were drawn up and ejected a given number of times. The syringes were then used for transferring sterile broth to sterile broth-bottles.

(3) As in (2), except that the syringes were dried after infection.

(4) As in (2) and (3), except that glass pipettes replaced the syringes.

Controls were done to check the technique.

The experiments, details of which were given, demonstrate that syringes cannot be disinfected by drawing up linseed oil, olive oil, or liquid paraffin five times, even at a temperature of 200° C., and confirm essentially the results of DREYER and AINLEY WALKER.

REFERENCES.

1. *The Technique of the Teat and Capillary Glass Tube.* Sir ALMROTH WRIGHT.
2. *The Resistance of Micro-organisms suspended in Glycerine or Oil to the Sterilising Action of Heat.* PATHOLOGICAL SOCIETY, July, 1912.
3. *B.M.J.* Aug. 2nd, 1919.

Mr. CECIL A. HOARE gave a demonstration on the occurrence of trypanosomiasis in the British sheep. He pointed out that a trypanosome in a British sheep was first observed by WOODCOCK in 1910. He only saw one trypanosome and, unfortunately, gave no description of it. In the following year a similar finding was recorded by BEHN in Germany. On *a priori* grounds WOODCOCK suggested that the trypanosomes in sheep were derived from the flagellates of the sheep-ked, *Melophagus ovinus*, which is a blood-sucking ectoparasite peculiar to sheep, and is nearly always infected with a flagellate known as *Crithidia melophagia*.

A fair number of works dealing with *C. melophagia* and its probable relation to trypanosomes in sheep have appeared, the majority treating this flagellate as a specific insect parasite. Thus, SWINGLE (1911) recorded a series of experiments which he had carried out with the object of establishing the relationship of the ked flagellate to a trypanosome of sheep. He was unable to infect sheep with the ked flagellate, nor did he find any evidence of trypanosomes occurring naturally in sheep.

Last summer Mr. HOARE started working on this question at the suggestion of Dr. C. M. WENYON. While this work was in progress, there came to hand several publications by NÖLLER, who, it appears, had been working on the same subject in Germany, and had already succeeded in finding trypanosomes in sheep by cultural methods.

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By using the same methods Mr. HOARE succeeded in cultivating the trypanosomes from sheep's blood, and, by subcultivating in different media, he has already kept cultures alive for more than a month.

In the primary blood cultures the trypanosomes appear after about eight days. They are then typically trypaniform in character, but the kinetoplasts in many already occupy an anteronuclear position; whilst a number show a posteronuclear arrangement of the kinetoplast as in true trypanosomes. In the subcultures the flagellates are of a typical Crithidia form and quite indistinguishable from those found in the sheep ked. All these types will be found under the microscopes.

The reason why so many investigators failed to find trypanosomes in sheep is accounted for by the very scanty number present in the blood. Mr. HOARE has examined a series of thick blood films from thirty-five sheep—a great number of which were proved by culture to be infected—without finding a single trypanosome.

There can be no doubt that the sheep become infected from the sheep-ked, which thus becomes the intermediate host of the sheep trypanosome, which now bears the name *Trypanosoma melophagium* (=*Crithidia melophagia*).

This trypanosome is, as far as is known, a harmless parasite, but it must be borne in mind that there are several diseases of unknown nature in sheep, which may prove to be due to this trypanosome.

The investigation is still in progress, and Mr. HOARE hopes to be able to test the question of transmission and pathogenicity later on.

The following preparations were shown :—

(1) Live trypanosomes from subculture.

(2, 3, 4) Stained preparations of trypanosomes from cultures.

(5, 6) Preparations showing gradation forms (from trypanosomes to crithidia).

(7, 8) Crithidia forms from the sheep (from culture).

(9, 10) Crithidia forms from the gut of the sheep-ked.

(11) Leishmania and pear-shaped forms from the rectum of the sheep-ked.

(12) Section of the gut of the sheep-ked showing flagellates attached to the wall.

(13) The intermediate host of the sheep-trypanosome, the sheep-ked (*Melophagus ovinus*).

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Dr. R. St. John Brooks showed the following preparations and cultures:—

(1) Microscopical demonstration of slide preparations of *Leptospira icteroides* and *Leptospira icterohæmorrhagiæ.*

(2) Cultures of micro-organisms illustrative of tropical bacteriology:—

Typhoid and paratyphoid fever: salmonellosis, including paratyphosus C, and various types of suipestifer.

Bacillary dysentery: Flexner types (including monkey strains) and Shiga.

Hæmorrhagic septicæmia group: *B. pestis*, *B. pseudotuberculosis rodentium* and Pasteurellas from rabbits, cattle, sheep and pigs.

Malta fever and abortion of cattle, including Feusier and Meyer's types of *B. abortus* and *B. melitensis.*

Acid-fast bacteria including various organisms isolated from leprous lesions, *B. tuberculosis* (human, bovine and avian) and Johne's bacillus.

Vibrios: V. choleræ, V. El Tor, V. para-cholera A and B (Mackie and Storer), and cholera-like vibrio from water.

Skin fungi: epidermophyton, trichophyton, etc,

Blastomyces: streptothrix, sporothrix, etc.

Blastomyces and torulæ from lesions from man and horses, *Coccidiodes immitis* from case of San Joaquin Valley disease (Coccidioidal granuloma), sporothrices from man and horses, streptothrix from camel, and Nocardia from eye disease, Khartum.

Eastern Fermentation Industries:—

Moulds from Saké, etc.: *Aspergillus oryzæ*, *Rhizopus Japonicus* and *Mucor* (*Amylomyces*) *Rouxii.*

Yeast from African millet beer (Pombé); Schizosaccharomyces (Pombé).

Yeast from Kefir: *Saccharomyces fragilis* and "Kefir" yeast (Hansen).

Sauces and pickles: *Saccharomyces farinosus* and *Aspergillus tamari.*

Dr. C. M. Wenyon gave a demonstration of preparations illustrating:—

(1) The structure of Trichomonas and its invasion of the tissues of the intestinal wall of human beings and rats.

(2) The hæmogregarine (*Hepatozoon canis*) of the leucocytes of the

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dog, showing the blood form, the schizogony stage in the spleen, and the oöcyst in the tick *Rhipicephalus sanguineus*.

(3) The hæmogregarine (*H. muris*) of the leucocytes of the rat, showing the blood form, the schizogony stage in the liver, and the oöcyst in the mite *Lelaps echidninus*.

Drs. J. G. THOMSON and A. ROBERTSON gave a demonstration illustrating the frequent occurrence of Charcot-Leyden crystals in the fæces of amœbic dysentery cases, and pointed out the value of these crystals as an aid to the diagnosis of such cases. The following specimens were shown:—

(1) A Charcot-Leyden crystal in the cytoplasm of a free *Entamœba histolytica*.

(2) Charcot-Leyden crystals from a sigmoidoscope scraping of an amœbic ulcer. Numerous small crystals arranged in clumps.

(3) Charcot-Leyden crystals from a sigmoidoscope scraping of an amœbic ulcer, showing several large crystals.

(4) Free *E. histolytica* along with Charcot-Leyden crystals in the same microscopic field from the blood and mucus of an amœbic infection.

(5) Charcot-Leyden crystals associated with the cysts of *E. histolytica*.

Nos. 1, 2, 3 and 5 stained with Heidenhain's Iron Hæmatoxylin; No. 4 stained with Ehrlich's Acid Hæmatoxylin and Eosin.

Microphotographs and camera-lucida drawings of Charcot-Leyden crystals in their relationship to *E. histolytica* were also shown.

Dr. A. CASTELLANI showed specimens illustrating the identification of sugars by the mycological method.

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(2) T.D.B. Vol 16 No 5 Nov 19

Proceedings of a meeting of the Society held on Friday, May 20th, 1921, at 8.45 p.m., at 11, Chandos Street, Cavendish Square, W. 1, Professor W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P. *(President)*, in the Chair.

A NEW METHOD OF TREATMENT OF TRYPANOSOMIASIS.

BY

CLAUDE H. MARSHALL, M.R.C.S., L.R.C.P., *S.M.O. Uganda Protectorate.*

I have very kindly been allowed to read to you to-night a paper on a new method of treatment of trypanosomiasis which has been originated by Dr. S. M. VASSALLO of the Uganda Medical Service and myself.

Sleeping sickness depends for its causation on three factors, namely, (1) the human host, (2) Glossina (tsetse-fly), and (3) the trypanosome; any two of these are harmless in the absence of the third, so that in dealing with this disease we have three different courses open to us:—

(*a*) Human beings may be excluded from areas infected by the Glossina;

(*b*) The fly may be driven away by depriving it of its shelter and food; and

(*c*) attempts may be made to cure infected persons.

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The first course (*a*) has been that most employed, and is the only one which has been generally successful in preventing the incidence of sleeping sickness. It amounts, however, to nothing more or less than surrender to the tsetse-fly, and, as a result, enormous areas of country have been evacuated and abandoned. (*b*) During the past ten years something has been done to stamp out the tsetse-fly by destroying its breeding-grounds, driving away its food supply, and cutting down the bush which forms its natural shelter. (*c*) Attempts to cure patients infected with the trypanosome have, in the vast majority of cases, proved unsuccessful, although individuals—certainly European and Asiatic, possibly also African—have recovered, but, taken generally, sleeping sickness must still be regarded as a fatal disease. Although many drugs cause a marked temporary improvement, each falls short of certainty in effecting a cure.

Most authorities are agreed that the administration of one dose of salvarsan, neo-salvarsan or atoxyl is sufficient to sterilise the blood-stream, but within a variable period, averaging about four months, the symptoms reappear, and the disease progresses to a fatal termination.

Excluding reinfection, two theories have been put forward by other workers to account for this, namely, (1) that the trypanosome assumes a resistant form and becomes what is known as "atoxyl fast," or (2) that some of the parasites are protected from the action of the drug by assuming a secluded position in the central nervous system.

That the second of these theories is the right one is, I think, proved by the following cases:—

Case 7.—A man with a few small glands in his neck was found to have trypanosomes in his gland-juice on June 1st, and again on July 1st, 1919. On July 10th, he was given an intravenous injection of 0·6 gms. of neo-kharsivan. On July 27th, his glands were smaller and harder, and no trypanosomes could be found in them; nevertheless, on October 28th, three and a half months after the injection of neo-kharsivan, living trypanosomes were found in the cerebro-spinal fluid.

Case 30.—A man with large typical chains of glands in the neck had numerous trypanosomes in the gland-juice on June 14th, 1920;

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0·6 gms of neo-kharsivan were injected into a vein, and three days later living trypanosomes were found in his cerebro-spinal fluid. Again, in Case 33 living trypanosomes were found in the cerebro-spinal fluid three days after an intravenous injection of neo-kharsivan.

In Case 34, there were trypanosomes in the gland juice on the 3rd August, 1920, and the man was given the usual intravenous injection of neo-kharsivan. Living trypanosomes were found in his cerebro-spinal fluid three months afterwards, though nothing abnormal could be found by repeated examination of his blood and gland-juice, either then or at the end of November.

In these four cases the blood and gland-juice were rendered free from parasites, presumably by the intravenous injection, but in both as early as three days, and as late as three and a half months afterwards, living trypanosomes were found in the cerebro-spinal fluid.

VASSALLO has found, in performing post-mortem examinations on one or two fatal cases of sleeping sickness, that the meninges of the brain are greatly thickened, and we suggest that the action of the trypanosome in the central nervous system is to cause such thickening of the membranes that the connection between the blood-stream and the cerebro-spinal fluid through the choroid plexus is occluded quite early in the disease, and that, as a result, drugs circulating in the blood-stream are not eliminated into the spinal fluid, so that when the trypanosomes in the blood are killed off by natural, or artificial means, the infection persists in the central nervous system, later on, may be, to reinfect the blood-stream again. On this point Professor KEITH has very kindly furnished me with the benefit of his views, and he states: "While the exact anatomy of the route is unknown, there is no doubt that infections in the basal cisterns do reach the cervical glands, and that very probably this occurs through the nerve-sheaths, where there is some connection between the cerebro-spinal and the lymphatic systems." On this assumption, the trypanosome is protected by its position in the body and its suggested resistance to atoxyl and similar preparations is not proved, inasmuch as the trypanosome would not be brought into actual contact with the drug.

In the treatment of sleeping sickness, therefore, two separate foci of infection must be considered: the initial infection of the blood-stream, causing fever, skin rashes, etc., from which the patient recovers, either

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with or without treatment, and the second focus in the cerebro-spinal fluid, where the trypanosomes cause cerebro-spinal lesions leading to the death of the patient.

Before the cerebro-spinal fluid has been invaded, there appears to be no reason why injections of arsenic and other preparations should not cure the patient; but once the invasion of the central nervous system has occurred, and drugs can no longer reach it from the blood-stream, all intravenous medication is useless, except to deal with such parasites as subsequently reappear in the blood-stream from the lymphatic system.

In the autumn of 1918, on the supposition that the above theory offered an explanation as to why all forms of treatment hitherto had proved so unsuccessful, I attempted intrathecal, in addition to intravenous medication.

My first case was a man with small typical glands (harbouring trypanosomes) in the neck, who was injected with 0·6 gms. of neo-kharsivan intravenously on September 26th, 1918. Three days later, 0·025 gms. of neo-kharsivan were injected into his spinal canal; he died the following day.

As this form of treatment failed it was not repeated, but the next case (a woman) was given an intravenous injection of neo-kharsivan, and three hours later two ounces of her blood were drawn off into a sterile covered vessel, which was left standing in a bowl of cold water for twenty hours, by which time the clot had separated. Twenty mimims of the clear serum were then injected into her spinal canal, after a corresponding amount of spinal fluid had been drawn off by lumbar puncture.

This woman was a well-marked case of sleeping sickness, trypanosomes being easily found in the large typical glands in the neck, and, though she has had no more treatment of any kind, and has been examined at intervals of three months, her blood has remained negative. When seen in December, 1920, twenty-seven months after treatment, she was quite well. Her blood and cerebro-spinal fluid were then negative, and the glands in the neck, which had completely disappeared soon after the injection, had not reappeared.

Thirteen other cases were treated in the same way. The amount of serum injected and the length of time between the injection of neo-kharsivan and the drawing off of the blood was varied. The results

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obtained up to the end of 1919 were published in the *British Medical Journal* of May 22nd, 1920.

In February, 1920, I was relieved by Dr. S. M. VASSALLO, and he took over the patients I had already treated, and, during the past year, has dealt with a number of new cases. The whole are shown in the table :—

TABLE.

Case Number.	Number of months since treatment to Dec., 1920.	Examined December, 1920: Blood.	C.F.S.	Glands.	
2	27	neg.	neg.	disappeared.	Quite well.
3	27				No. records.
4	24	do.	do.	do.	Quite well.
5	18				No records.
7	19	do.	do.	do.	Quite well.
8	16	do.	do.	do.	do.
9					Died; very advanced case.
10					do. do. do.
11	13	do.	do.	do.	Quite well.
12	13	do.	do.	do.	do.
13	13	do.	do.	do.	do.
14	13	do.	do.	one gland neg.	Well.
15	12	do.	do.	disappeared.	Quite well.
16	8	do.	do.	do.	do.
17	8	do.	do.	do.	do.
18	8	do.	do.	do.	do.
19	8	do.	do.	do.	do.
20	8				Not reported for examination. No records.
21	8	do.	do.	do.	do. do. do.
22	8	do.	do.	do.	
23	8	do.	do.	do.	Treated atoxyl; serum; quite well.
24	8	do.	do.	do.	do. do. do.
25	8	do.	do.	do.	do. do. do.
26	7½	do.	do.	do.	Quite well.
27	7½	do.	do.	do.	do.
28	7½	do.	do.	do.	do.
29	7½	do.	do.	do.	do.
30	6½	do.	do.	do.	do.
31	6½	do.	do.	do.	Treated plain serum. Quite well.
32	6½	do.	do.	do.	Quite well.
33					Died after anæsthetic—delayed chloroform poisoning.
34-56	Under six months since treatment and not yet re-examined.				

In this connection I must explain that all work done by both VASSALLO and myself on the subject of sleeping sickness has been in addition to our ordinary duties as District Medical Officers, and, as a

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result, several of our records are incomplete. For instance, several cases on the table are marked as "no records." This was owing to the fact that early in 1919 I had to leave my station for over two months to undertake other duties, and during this period a severe famine occurred in the district, causing the death of between 20,000 and 30,000 natives. On my return I could learn nothing of Cases 3 and 5, except that they were reported to have died, but no definite information could be obtained.

Again, Case 10 was discharged to his home and later on was reported to have died. Since he was in an advanced stage of the disease we have presumed that he died of sleeping sickness.

Case 6, omitted from the table, was a dog infected with the *T. gambiense*, or human strain of trypanosome, and was treated as a control to see if an intravenous injection of neo-kharsivan alone would cure the disease, but the animal had to be shot about a month later as being hopelessly ill.

Fifty-six cases are recorded, and fifty-two of these have been treated by some form of serum.

Of these fifty-two, six are dead or presumed dead; practically all the others were seen in December, 1920, and found to be perfectly well. In all cases the blood and cerebro-spinal fluid were examined, with negative results.

As a point of considerable interest, although all these cases originally had well-marked glands, yet after serum treatment these glands have invariably disappeared completely, while in cases not treated by serum the glands remain.

Of the six patients who have died, two disappeared during the famine, and one, at any rate, of these is stated to have died of starvation. Two died of intercurrent disease and two we presume died of sleeping sickness. These last, before they came for treatment, were very advanced cases of the disease, one being unable even to stand.

On the other hand, one case, the woman referred to, has remained quite well after twenty-seven months; another after twenty-four months, and seven more after over a year since being treated by a single dose of serum.

One patient treated in April, 1920, aged about 60, cycled in thirty

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miles one morning for re-examination, a notable feat for him, since he stated that he had not been able to ride a bicycle for at least a year before treatment. Three other cases, treated over a year ago, when they admitted to being impotent—indicating involvement of lumbar nerve centres—are no longer so. They have since become fathers. Enough has been said to show that this method of treatment is successful, or, at any rate, certainly gives better results than have hitherto been obtained by other methods of treatment, especially when it is borne in mind that all these cases have had only one dose of serum.

The question, then, arises whether this injection of salvarsanised serum into the spinal canal has given such good results from its antiseptic, or rather antiparasitic, action alone. It appears to us as improbable, if 0·6 gms of neo-kharsivan are dissolved in all the blood in the body, and then 20 minims of this mixture are again diluted by the whole bulk of the cerebro-spinal fluid, that the resulting solution can be powerful enough to kill off the trypanosome.

We suggest that some trypanolytic antibody is normally formed in the blood as a reaction to the invasion by the parasite, and that this effects a natural cure as far as the blood-stream only is concerned. However, since in fact the cerebro-spinal fluid contains no antibodies, opsonins, or alexins, and as we suggest the cerebro-spinal fluid is cut off from the blood-stream by the disease, it follows that a natural cure cannot take in the central nervous system, but injection of blood serum containing the trypanolysin into the spinal canal is sufficient to kill the parasite, either directly or, as Dr. ABADIE of Paris believes, by stimulating the formation of further complements.

To support this theory of trypanolysins we have in several cases given the patient no intravenous injection at all, but have simply drawn off a quantity of blood from a vein (under aseptic conditions) and allowed it to stand for as long as three days; we then injected the serum into the spinal canal, expecting that any contained trypanosomes would have died from exposure and the trypanolysins in the serum would effect the cure of the infection in the cerebro-spinal fluid, at the same time the parasites in the blood-stream would be destroyed by their own antibodies.

The first case so treated (No. 31 male, *æt.* about 30) has now

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gone on well for six months up to December, 1920. No trypanosomes could then be found in his blood or spinal fluid; the glands in his neck had disappeared.

Following up this line of treatment, VASSALLO is trying now the effect of the intraspinal injection of serum from a previously treated case into a patient with sleeping sickness. It is, however, yet too soon to form any opinion as to its effect.

Of more interest in this connection is the case of Mr. J. G. Rubie, our only European patient, who has been good enough to come here to-night. He had attacks of what he believed to be malaria, commencing July 30th, 1920, and, finally, had his blood examined on August 24th, when it was found to be swarming with trypanosomes.

I first saw him as a patient on September 24th, 1920. He had general lassitude, malaise, distaste for food, attacks of vomiting, with pains in the back and limbs, and a dull headache. He had lost two stone in weight. There was one small gland in the neck (which may possibly have been septic in origin) and his blood showed two or three trypanosomes in each field.

He was given 0·9 gms. of neo-kharsivan intravenously and 12 c.c. of serum into the spinal canal, the blood being drawn off after half an hour. He showed a very severe reaction for several days and then made uninterrupted recovery. He has lost all symptoms, regained his lost weight, and will, I think, tell you that he feels absolutely well. It is a year since the probable date of infection, and nearly eight months since this single treatment which I have just detailed.

However, on November 2nd, 1920, about six weeks after the injection of serum, while nothing abnormal could be found in his blood, sub-inoculation on that day into two rats and a monkey resulted in one rat showing trypanosomes on November 30th, while the other rat was negative. The monkey became very sick though it did not die, and no trypanosomes where found in its blood.

At this time, then, Mr. Rubie still harboured the parasite in his system though apparently a strain of a low virulence, since the monkey showed only a mild infection.

On December 21st, 1920, the blood was examined again, both fresh and stained, by Professor J. EYRE, who reported one degenerate trypanosome without a nucleus, the only organism found in eight slides. Sub-

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inoculations into two guinea-pigs were negative. One of these guinea-pigs was post-mortemed about six weeks later, and was found absolutely healthy.

Then there arose the question as to what had occurred to account for this history, and three possibilities were considered :—

(1) An error in technique, that is to say, that the dose of neo-kharsivan had been too large or the time between the injection and the drawing off of the blood too short. Professor EYRE suggested that a provocative dose of 0·3 gms. of neo-kharsivan, and a period of one and a half hours before drawing off the blood, would establish the most favourable conditions for the production of trypanolysins, and

(2) That treatment had been commenced too early in the disease, and the body had not had time to react and produce antibodies to the trypanosome, with the result that the serum was valueless. If this were the case, it would, I imagine, be incompatible with the experience of most of us that late rather than early treatment in a progressive disease could be to the patient's advantage, and

(3) That the presence of degenerating trypanosomes in the body after treatment was merely a feature of the normal process of the cure by this method. We accept this as the explanation.

It was decided, in consultation with Dr. PROUT and Professor EYRE, to examine Rubie again, and, if no trypanosomes could be found, to allow matters to take their course without any attempt at further treatment. So, on January 20th, 1921, lumbar puncture was performed and the cerebro-spinal fluid was examined, both fresh and stained, also 1 c.c. each was sub-inoculated into two rats ; all results proved negative and the cerebro-spinal fluid contained no cells of any kind.

The patient was examined again on April 7th, 1921, and his blood found to be negative on examination of fresh and stained slides and sub-inoculation into two rats and six mice. On May 2nd his cerebro-spinal fluid was normal and negative on subinoculation into three rats and two mice.

That Rubie's presumed cure is not due to the initial dose of neo-kharsivan killing off all the trypanosomes in the body is certain, since the sub-inoculation on November 2nd showed that he still harboured the parasite; that treatment was effective, both in technique and time, is shown by his excellent clinical history and the completely negative

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results of microscopical examination and of further sub-inoculations since the beginning of this year. We believe that the explanation of his history is that the injection into the spinal canal dealt with that focus of infection while the continued formation of trypanolysins in the blood gradually destroyed the parasites in the blood-stream.

Our two most advanced cases of sleeping sickness did not prove amenable to treatment, and we are proposing to treat such cases in the future by the injection of serum into the lateral ventricles as well as into the spinal canal, on the supposition that something of the nature of an obstructive hydrocephalus occurs in these patients, through the occlusion of the foramen of Majendie by thickening of the membranes, and that, as a result, intrathecal injections are unable to reach the cavity of the brain. This idea is supported by the fact that often, in advanced cases, no cerebro-spinal fluid at all can be obtained by the lumbar puncture.

We intend, however, to investigate this point by injection of indigo-carmine into the lateral ventricle and endeavouring to recover it by lumbar puncture. Inability to do so would prove the correctness of this theory, and indicate that these cases require serum to be injected directly into the brain cavity.

To sum up, as far as our experiments have gone, they show (1) that there are two distinct foci of infection in an established case of sleeping sickness, and that of these the focus in the central nervous system is the important one, but that this can be dealt with effectively by a single dose of some sort of serum; (2) that the results of such serum treatment in over fifty cases appear to be better than any of the results hitherto obtained by repeated intravenous or subcutaneous injection of salvarsan, atoxyl, antimony or other preparations; and we suggest that the curative effect results from the formation and action of some form of trypanolysin.

If our theories are correct, there appears no reason why animal serum should not be used for treatment, why some degree of prophylaxis and immunity to trypanosomiasis should not be obtained, and why early diagnosis should not be effected by some form of complement fixation test. Upon all these questions VASSALLO is carrying out experiments.

Further, the line of treatment adopted for human beings can probably be applied to animals. The cure of "fly" disease in horses and cattle should prove of great economic value, and, given time and oppor-

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tunity, both VASSALLO and I have great hopes of being able to deal simply and effectively with trypanosome diseases both in man and beast. But we are very anxious that we should be assisted by the Veterinary branch of Medical Science in these researches. The problem of trypanosome infection is the same whether applied to human beings or other animals, and experiments on the treatment of each dovetail into one another, so that the co-operation of a veterinary officer in future work is extremely desirable.

DISCUSSION.

The PRESIDENT: This is the second paper we have had on trypanosomiasis this session. The first was by Professor WARRINGTON YORKE, who raised a very lively discussion in this room because of his criticism of the proposal to spend £30,000 on trypanosomiasis investigation from the zoological side. It was felt at the time that a research of this kind would be extremely valuable, but that it was somewhat limited in scope, and that there were other aspects of the question which required attention; for instance, there were the medical, the veterinary, the laboratory, the administrative and other sides. To-night we have had the medical side put to us, and it is a very important one. I consider that this paper is one of the most important that have been placed before this Society. If the results which have been told us to-night are maintained in the future, and are confirmed by further investigations, they indicate, to my mind, a new and brilliant era in the treatment and prevention of trypanosomiasis.

What does that mean to Africa? I have only to bring to your minds the vast area of Africa infested with the tsetse-fly, the scourge which trypanosomiasis has been to the African population, and the ever-present danger of this disease spreading. These conditions present a great and perplexing problem, and I think that any researches that may be placed before this Society which in any way contribute to a solution of this subject are particlarly welcome. I place in this category the paper which has just been read. These experimental observations of Dr. VASSALLO and Dr. MARSHALL have not been made on the lower animals, but they have been on men and women, and the results, so far as they go, certainly appear to me to hold out a reasonable prospect of a reduction in the mortality and the dangers of, at least, one of the forms of trypanosomiasis.

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In presenting his statement to us, Dr. MARSHALL has, very rightly, excluded the twenty-three cases that were treated only six months previously. This leaves thirty-one cases, and one of these thirty-one died under chloroform, so that it can hardly be counted. If we take this group of thirty cases which has been treated by the serum only once, and more than six months ago, we find that twenty-two cases are still alive and well; that is to say, that 73 per cent. of the cases that have been so treated are alive and well after six and a half months and more. If we analyse Dr. MARSHALL'S table further, we shall find that thirteen cases were treated a year and more ago, and that eight of these, *i.e.*, 61 per cent., are alive and well. These percentages I have placed at the very lowest estimate, because there were four cases about which there was no information obtainable, and, for the purposes of these percentages, I have treated them as persons who died of sleeping sickness. And there is another, No. 14, in regard to which it is stated that the cerebro-spinal fluid and the blood were all right, and that there was only one gland negative; and I understand from this statement that there were some glands that were positive.

Dr. MARSHALL: One palpable gland, but it was negative; no other glands were enlarged.

The PRESIDENT: That being so, instead of eight being alive and well there were nine alive and well; in other words, 69 per cent. of the cases are alive and well at the present moment. I think that to have treated such a series of patients suffering from trypanosomiasis with the success which these percentages represent is a very great and remarkable achievement. And it is the more remarkable when we remember that hitherto the successes have been comparatively few by the other forms of treatment; and it is pretty well recognised that when a case is treated in the endemic area it is pretty well hopeless.

Now it remains to be seen whether the same results can be obtained with the Rhodesian type. I see no reason why it should not, because I look upon this treatment as being based on a scientific foundation; there is using the serum separately on the cerebro-spinal fluid and on the circulating blood, and there is also the preparation of this serum by letting it stand and allowing the products of the trypanosomes to die and excrete their intracellular and extracellular enzymes, or whatever you

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may choose to call them. In treating the cerebro-spinal fluid and the circulating blood as different systems the authors are absolutely correct; they are correct in so far that no metallic medicament injected into the blood-stream reaches the cerebro-spinal system. Professor HALLIBURTON, whom we are glad to see here to-night, in his presidential address to the Neurological Section of the Royal Society of Medicine, in October, 1916, treated upon this subject, and showed that this was the case in normal individuals. Perhaps we may have the advantage of hearing him explain this. I am in favour of the trypanolysin theory, because, after all, the treatment which has been advocated just now is very similar in method to that which is used for the curative and preventive treatment by vaccines and antitoxins. It does not matter whether you are dealing with the vegetable world, such as bacteria, or with the animal world, as you are in the case of trypanosomiasis, the enzymes, or the antitoxins, whatever they are, appear to have practically the same action—they have an activating effect on the cells and tissues of the body and render them capable of destroying the parasites. This autoserum therapy is very interesting, and is receiving attention of late. Recently, in the *Indian Medical Gazette*, there is an article by Dr. SENN, of Bombay—an article under the supervision of one of the medical men of the Indian Medical Service— in which some very instructive cases are given of treatment by this serum therapy. In the case of patients who are suffering from certain illnesses, he draws the blood and dilutes it with distilled water, in order that the ferments shall get into the surrounding fluid, and then he injects into the patient this fluid, and some of the results reported are very remarkable.

You will have gathered from what I have said that I am very much impressed with the value of this new treatment, and I do hope that some movement will be inaugurated for putting it to the test, not on a small scale but on a large scale. And I agree with Dr. MARSHALL that the veterinarian should come in and assist. But I also think that other scientific men should assist. And I would like to see some of the wealthy men coming forward and helping financially with it. I would like to see a Rockefeller assisting. We want some British Rockefeller very badly to encourage the application of scientific discoveries to our tropical possessions.

You may think I am taking a very rosy view of the researches of

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Drs. MARSHALL and VASSALLO, but it is now over twenty-five years since HAFFKINE and myself worked out successfully on men and women the problem of anticholera inoculation, and we began with a much less promising prospect than in the present case. You know that anticholera inoculation has been the foundation of many of the present-day vaccines.

I am sure this paper will give rise to a very interesting discussion, and I hope you will speak freely and criticise the paper, because it is by criticism that people learn. We would like first to hear Professor HALLIBURTON on the cerebro-spinal system.

Professor W. D. HALLIBURTON, F.R.S.: The particular point I drew attention to in the presidential address you have alluded to, before the Neurological Section of the Royal Society of Medicine, the part which bears upon what we are dealing with to-night, is with regard to the normal formation of the cerebro-spinal fluid and its normal function. The cerebro-spinal fluid is a secretion; it is not a leakage fluid, like lymph is from the ordinary blood-stream. If we may compare a salivary gland to the particular mechanism which forms this fluid, I can represent the difference between them in a very simple way on the blackboard. In the salivary gland there are groups of cells, arranged in acini, and each acinus consists of a number of large cells which border the space called the lumen, whence the saliva passes through the salivary ducts. Each of these acini is surrounded by blood-vessels. The blood leaks, and we get lymph formed. Saliva has a composition of its own, and it is of fairly constant composition. So from the blood the exudation takes place to the lymph, and the cells work up the lymph into the new material called saliva, and, ultimately, we get the saliva poured out, at considerable pressure. In the case of the choroid plexus, which Sir FREDERICK MOTT has called the choroid gland, you will find something of the same sort; but in this case the blood-vessels are inside, and they are coated over by cubical cells, which are true secreting cells, and show histological changes similar to those which can be detected in various stages of secretion in the salivary gland. The blood leaks, and gives rise to lymph; these cells take up from the lymph the materials which they want. The direction again is from blood to lymph, then into the cells, which then pour their secretion into the cerebro-spinal space. Just as the saliva is of fairly constant composition, so the fluid which is formed here (see diagram),

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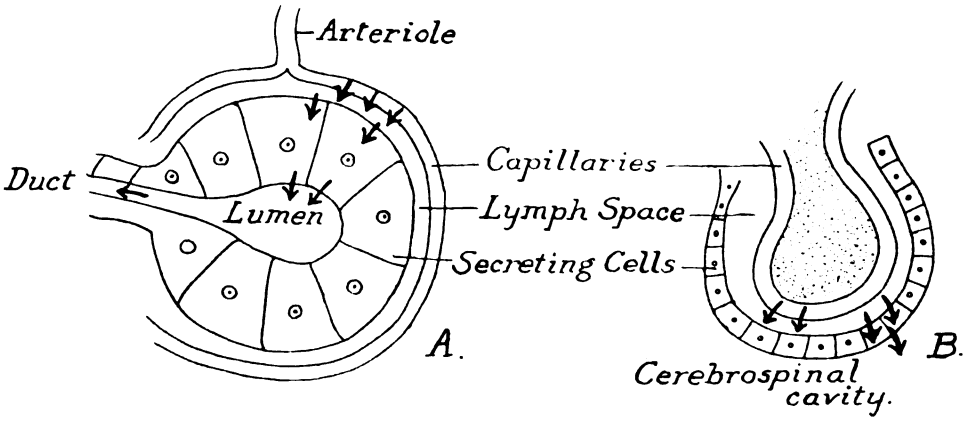

and enters the cerebral ventricles and the other spaces, is of constant composition. It is especially important that it should be constant, because that fluid ultimately penetrates to the spaces which surround the nerve cells in the central nervous system. These cells are very sensitive, so that any error in composition of that fluid would produce serious effects on the nerve cells. The cerebro-spinal fluid is sometimes called the lymph of the brain. That is a very slipshod expression. It is contained in channels which resemble lymph channels, but in the case of the lymph there is always the possibility of the passage of the fluid and other materials in both directions, from the blood to the lymph, and from the lymph to the blood. But in the case of cerebro-spinal fluid there is a passage in one direction only; this ideal saline fluid maintains the normal osmotic pressure necessary for the life of the nerve cells; the cells which secrete the fluid take good care that nothing unusual comes through. They form what we may call a barrier to prevent harmful materials from passing into the cerebro-spinal fluid. It is quite true that materials introduced into the cerebro-spinal fluid can get into the blood-stream, but, except for the choroid secretion itself, there is no passage (on account of the guarding action of these choroidal cells) of poisonous materials like arsenic or antimony from the blood. In disease these choroidal cells still maintain their rôle of a stalwart barrier, and foreign materials circulating in the blood are kept back, only the normal fluid coming through. There are one or two very volatile materials which get through, and of these alcohol is one; it is the only one about which we are quite certain. Nature lets alcohol through; hence harmful effects on the nerve cells are produced with comparative

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ease by this poison, but metallic poisons are kept back, even in disease. When, in syphilis, spirochætes (or, in sleeping-sickness, trypanosomes) get in by a back door, they have reached a harbour of refuge, they are extra-vascular, and the only way to attack them is, not to inject anything into the blood-stream, because it will not get into the cerebro-spinal fluid, but inject it straight into the cerebro-spinal fluid. That has been tried in the later manifestations of syphilis, such as locomotor ataxy, but the result is similar to that which Dr. MARSHALL has recorded this evening; you not only by this means kill the parasite in its lair, but you also kill the host which harbours it, and the treatment is a good deal worse than the disease itself. Therefore we must be extremely thankful to Dr. MARSHALL and his colleague for finding material which is fatal to the parasite but which is not fatal to the man who harbours it. I think, therefore, that in the explanation which they have advanced on page 12, they have slipped into a misapprehension of what is really the case. They suggest that the action of the trypanosome in the central nervous system is to cause such thickening of the membranes that the connection between the blood-stream and the cerebro-spinal fluid through the choroid plexus is occluded quite early in the disease, and that, as a result, drugs circulating into the blood-stream are not eliminated into the spinal fluid. What occurs in the disease is really on all fours with what occurs in health. The choroidal cells are still healthy, for if they were diseased, they could not maintain their usual office of stalwart guardians and prevent any foreign material passing into the cerebro-spinal fluid. I have very great respect for the encyclopædic knowledge of Sir ARTHUR KEITH, but I think he has gone wrong in what he is quoted to have said in a later part of the same paragraph. It is true that the exact anatomy of the route is unknown; but, he goes on, "there is no doubt that infections in the basal cisterns do reach the cervical glands, and that very probably this occurs through the nerve sheaths." That statement is founded on the writings of a number of French observers a good many years ago. But within recent years my friend, Professor DIXON, and myself, have reinvestigated the matter, and we found that materials introduced artificially into the cerebro-spinal canal did not leave by lymph channels. If you inject material which you could easily recognise, such as methylene blue, no trace of blue passes into the lymph stream; the lymph collected from the thoracic duct

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contains none; the blue lines which one would see along the nerves if the lymphatics were filled with blue fluid, are invisible. What does occur is, that the fluid which is being continually formed does not find an exit by the nerves or their lymphatics, but through the microscopic arachnoid villi which project into the venous sinuses mainly at the base of the brain. Within a few seconds after the injection of such a material as methylene blue in the cerebro-spinal canal, although you can find no trace of it in the lymph, you can find it in the blood, and, in a few minutes, in the urine. Communication is not via the lymph-stream, but via the blood-stream, and the arachnoid villi—which WEED and his colleagues in America have described—are now regarded as proved to be the sites of the leakage of the excess of cerebro-spinal fluid back into the blood-stream, but not into the lymphatic system.

Dr. P. MANSON-BAHR: In medical practice in London we have not, of course, access to as many cases of trypanosomiasis, as Dr. MARSHALL has had in Uganda. Nevertheless, from the few cases I have had to treat I have been able to draw some conclusions. The first question, as bearing upon the points under discussion to-night, which I would like to ask is this: Are we quite certain that the invasion of the cerebro-spinal fluid by trypanosomes is necessary in order to produce the sleeping-sickness state? I maintain we are not. I have one case in my wards at present who was infected originally on the Congo in 1916; he had an acute attack of trypanosome fever, typical lymphatic glandular enlargement, with a typical rash, but trypanosomes have been found on one occasion only—in 1919, three years after the onset of the disease. The cerebro-spinal fluid has been repeatedly examined and his blood injected into animals consistently with a negative result. Yet that man to-day, five years after his original infection, presents a typical clinical picture of the sleeping-sickness state. He lies prostrate in bed, with incontinence of both urine and fæces, and a mental degeneration of a severe degree. We have, therefore, no right to say that the sleeping sickness state is invariably caused by a trypanosome infection of the cerebro-spinal fluid!

Another case is one of an officer, also from the Congo, infected early in 1919, who has been under my care for a year. Everything possible in his case has been tried, including Dr. MARSHALL'S treatment. Intravenous injection of salvarsan in doses of 0·6 to 0·8 grammes has

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made no difference in the numbers of trypanosomes circulating in his blood. Therefore, in view of this, and in view of experiments upon laboratory animals, I cannot see that there is any justification for assuming that this drug possesses any trypanolytic action, and therefore I cannot understand the contention that salvarsanized serum in trypanosomiasis can have any action whatsoever. Another point to be taken into consideration is the habit which trypanosomes, in this case of *gambiense* type, have of appearing in the blood-stream in a periodic manner. In this case, in spite of atoxyl, antimony tartrate, salvarsan, and other drugs, the trypanosomes appear with regularity about every three weeks; they have been very numerous indeed, numbering as many as 30 to every 200 leucocytes. In this case too, the cerebro-spinal fluid is free from trypanosomes. I carried out the injection of salvarsanized serum, but, beyond making the patient very ill and giving him an intense headache for a week, it had no effect whatsoever upon the circulating trypanosomes.

Therefore, whilst congratulating Dr. MARSHALL on the splendid results of the treatment in his hands, we must look upon this problem in the true scientific spirit and confess that at present there is no specific treatment for trypanosomiasis. I have not had any good results by employing the same methods, nor can I see how the injection of such a small amount of practically normal human serum can have on such a resistant organism!

In any case, I should like to ask him how I have gone wrong in my technique and, in doing so, I would express my very real anxiety for the two cases at present under my care.

In estimating the exact value of the undoubted recoveries from trypanosomiasis (*T. gambiense*)—and Sir PATRICK MANSON and others record some sixteen—we must remember that, as in the case of other protozoal infections, malaria, for example, there is no reason why trypanosomiasis should not be a self-limited disease. Why is it that the malaria parasite (*Laverania malariæ*) kills some people in twenty-four hours and in others produces no symptoms at all?

Several of Sir PATRICK'S trypanosome cases were cured before the days of intravenous medication; they were cured by intramuscular atoxyl and even liquor arsenicalis by the mouth, yet we are not at present blazoning forth either of these two drugs as wonderful

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discoveries, specific for the trypanosome, or as opening up a new future for Central Africa, which has actually been stated to-night.

Dr. G. C. Low: The subject of the treatment of trypanosomiasis is a difficult one, and I do not wish, by any criticisms I make now, in any way to damp the enthusiasm of the younger generation in trying to devise better lines of therapy for this serious malady. I think I am specially competent to speak upon this subject, as I began my studies upon it as long ago as 1902 in Uganda, with the first Sleeping Sickness Commission. I saw and studied many cases there, and since that date have had the opportunity of seeing all of Sir PATRICK MANSON'S and Dr. DANIEL'S cases treated here in England, while, in addition, I have had a considerable series of my own. As one gets older one becomes more dubious as to how far one can definitely say any given protozoal disease is really cured. Take syphilis, for example, a disease due to a treponema, and, although not a protozoal one, akin in many ways to trypanosome infections. When salvarsan was produced, one dose was said to cure anyone, but now we know that even ten doses, plus mercurial treatment as well, fail, in many instances, to bring about the desired result. I would like to ask Professor HALLIBURTON one question: I had an idea that potassium iodide could reach the cerebro-spinal fluid when given by the mouth. Is that so?

Professor HALLIBURTON: Some people say so; I have tried it but failed.

Dr. Low: Well, then, let us admit that it is difficult or impossible for some drugs to reach the cerebro-spinal fluid, and so to reach the trypanosomes, if they lie up there. What proof, however, have we that that is the only place the trypanosomes are resting in? How do we know they do not lie up in the spleen, bone-marrow, or other tissues of the body? If that is so, we have to return to the idea that some of the parasites become resistant against the drugs used, an analogy present in malaria and other protozoal diseases. There are some strains of malaria that are very resistant to quinine, just in the same way as certain strains of the Leishman-Donovan parasite resist antimony. I have a patient now with a leishmanial sore on his nose which has resisted 34 grs. of antimony intravenously, plus full courses of X-ray treatment as well. Even with all

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this, parasites can still be cultivated from the lesion. Dr. MANSON-BAHR has told you of similar resistant trypanosomiasis cases to-night, and yet one of these had had Dr. MARSHALL'S exact treatment. One of these cases I saw (for Dr. DANIELS) when the patient first arrived in England, some years ago, and yet, though only a Nigerian case—the type more easily cured than any other—and though having all the different recognised treatments, he is now dying of typical sleeping sickness. I have another similar case under my own care now. Then, again, take the Rhodesian cases; all who have come home to England have died with the exception of one, and he had to have somewhere about 270 grs. of antimony to effect a cure, if he really is cured. Another of these cases lived over two years. He was kept under constant observation during the whole time, and, though in the middle of his time he appeared quite cured and healthy, he finally died, and at the autopsy the typical lesions of sleeping sickness were found. STEVENSON has recently shown that the trypanosome is not so much a blood parasite as was at one time thought; it can escape from the vessels and be found in the general tissues of the brain and other parts. How, then, if it is lying quiescent in the brain tissue, can injections into the spinal canal reach it? The perivascular small-celled infiltration, though well seen round the brain capillaries, is not limited to these areas, but occurs around the small vessels of the heart and generally throughout the body. It is, without doubt, produced by the irritation of the toxins given off by the parasites generally distributed throughout the body.

With regard to Dr. MARSHALL'S table, one must not forget that cases of trypanosomiasis may remain latent for long periods of time. I remember such a case before going to Uganda, that of a boy who died of sleeping sickness in Wales years after leaving the endemic tropical area. Bearing in mind such cases, one must not jump to the conclusion that all Dr. MARSHALL'S patients, even though some of them have apparently been well for thirteen months, are cured. It is rash to conclude that anyone is really cured under three, or one might be safer in saying five years. Some of the old 1902-1903 cases are, or were recently, alive, and one can of course definitely speak of them as having recovered. When I was in Uganda, the epidemic was at its height, and patients were dying in three or four months' time as an average, but even so, some were of a chronic type and lived a year or longer. It is almost

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certain, as far as Uganda is concerned, that the disease must have lost this extreme virulence at the present day, and this may easily explain the mildness of Dr. MARSHALL'S cases.

As regards the statement that the meninges are greatly thickened, this is not so. I have done a very large number of autopsies on such cases, and the great majority show nothing macroscopically at all, apart from a slight ground-glass-like appearance of the cortex of the brain. Further, sections confirm this, the small celled infiltration under the meninges being only found in a very thin layer. When the meninges were grossly thickened, secondary infections with organisms were always found.

Let us pass now to the case of the trypanolysin or trypanolytic antibody. Though not denying that some such substance may exist, it does not appear that it can have much potency. I have never heard of any case of natural cure in a white person. Such cases invariably end fatally at varying periods after infection. If the antibody is so potent that the amount contained in twenty drops of serum injected into the spinal-canal can completely sterilise the patient of his whole infection, why cannot it do so when circulating in the blood? Dr. MARSHALL may reply, the antibody cannot get into the cerebro-spinal fluid; but how can he prove this? And besides, as I have already shown, the idea that the trypanosomes are only present in the cerebro-spinal fluid in sleeping sickness is begging the question.

I do not know if Dr. MARSHALL is aware that an exactly similar theory was put forward for para-syphilitic conditions, such as general paralysis of the insane and locomotor ataxia, the idea similarly being that the treponema was locked up in the nervous tissues and that drugs given by the blood could not reach them. For this, salvarsanized serum was injected into the spinal canal, and even into the lateral ventricles, with, I believe I am correct in stating, absolutely negative results. If so, then I personally cannot see how a similar treatment will succeed in trypanosomiasis when it has failed in syphilis.

One does not like, as I have already said, to damp enthusiasm or to throw cold water on any new work. No one would be more pleased than myself to know that we had at last a definite cure for trypanosomiasis and its late sequel, sleeping sickness; but in this case I must say distinctly that the verdict is "not proven." Till we have further work accurately

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and scientifically carried out, it is premature to talk of this treatment as a cure, and it is not fair, by making such statements, to build up the hopes of those unfortunate individuals who happen to be suffering from the disease.

I honestly cannot see how injecting a few doses of the patient's own serum into his cerebro-spinal fluid is going to cure him. Nor can I believe that one injection of salvarsan will cure any case of trypanosomiasis. (*Vide* remarks on syphilis.)

Take the case even of the gentleman who has attended to-night. Six weeks after the treatment, trypanosomes were found in his blood, and since that inoculated animals have become infected. How can one talk then of this man being cured, even though no trypanosomes are present at the moment, especially after having heard from Dr. MANSON-BAHR of his case, where, though no trypanosomes have been found for a long time in the cerebro-spinal fluid nor in inoculated animals, yet the man is dying of sleeping sickness.

Lieut.-Col. R. H. ELLIOT: May I intervene in order to give a new turn to the discussion? A number of us are very greatly interested in Dr. MARSHALL's work, and we feel that that work ought to be given an opportunity for further continuation. We, therefore, think that whether he is right or wrong—and many of us do not feel competent to discuss that subject on the higher planes—this plan of treatment should be given a very full trial. I would further suggest that, if possible, some report should be made to the Colonial Office by this meeting, with a view to facilities being afforded for the investigation of the method of treatment now advocated, not merely from the point of view of human trypanosomiasis, but also from that of animal trypanosomiasis. We, many of us, know the tremendous amount of thought and anxiety which trypanosomiasis is giving to the Government of Uganda at the present time. I am in frequent touch with one who is engaged on work in connection with sleeping sickness, and I, therefore, feel a very personal interest in the subject. If this new line of treatment is right, there are enormous advantages to be gained from it, and the commission of investigation should not work at a disadvantage. The disadvantages of trying the new method of treatment, even if it prove useless, are negligible, but the advantages, if it is right, are very great. I am in a position to say that

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some of us have reason to believe that if any such movement is put on foot, we should not find it very difficult to put our hand on a good deal of money for the purpose.

Dr. E. T. JENSEN: Colonel ELLIOT said money would be forthcoming for this. I think I am within the facts when I say a substantial sum has been promised if this scheme is approved by the tropical diseases section of the medical profession. Will you ask, sir, whether this meeting approves of Dr. MARSHALL'S treatment being tried out?

Lieut.-Col. ELLIOT: I second that proposal, that every facility be given for Dr. MARSHALL'S work to be carried further, with a view to it being thoroughly tested.

This was put to the meeting and carried *nem. con.**

Sir HUGH CLIFFORD said that even if only a small number of cures could be effected by Dr. MARSHALL'S method, experiments to test it should be systematically carried out. There were many Medical Officers on the West African Medical Staff competent to take charge of such researches, and he would urge the Colonial Office that such experiments should be made in Nigeria, for which his Government would not grudge a few thousand pounds.

Dr. W. T. PROUT: I shall not say much about the technical part of this paper, except that I do not take quite such a pessimistic view of the matter as do Dr. LOW and Dr. MANSON-BAHR. I do not think Dr. MARSHALL has gone so far as to make a definite claim that this is a cure; he has simply said the indications, so far, are very hopeful; that this is a method which has afforded relief in a considerable number of cases for a considerable period; and, I think, we may hope that this new line which is being carried out will prove fruitful. I think the facts which have been put forward are sufficient to justify us in encouraging him in every way to carry out further investigations. So far as the patient who is here to-night is concerned, I have seen him since he returned to this country in December last, and his improvement has been progressive. It will be agreed that to-night he looks even aggressively healthy, and the last report of Professor EYRE was that he

* This resolution was *ultra vires* as previous notice had not been given.

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did a lumbar puncture and there was no increase of pressure; he inoculated two mice and one rat, and they were unaffected, the cerebro-spinal fluid has been centrifuged, and the blood examined carefully, and it has been found negative. There has been no repetition of these periodic outbursts while he has been in this country, and there are very strong grounds for believing he has got rid of these parasites. I do not think the analogy with malaria which has been made is quite a fair one. We know it is self-limiting in a few cases, that persons lose the disease without treatment; improved conditions of life enable them to get rid of the parasite. I do not know of any case in which trypanosomes have been swarming in the blood and the disease has proved to be self-limiting.

With regard to the question of a further investigation, I shall have an opportunity of putting the matter before the Colonial Office, and shall support the scheme in every possible way.

Dr. MANSON-BAHR: May I say a few words in explanation of Dr. PROUT'S dictum upon the pessimism of the physicians of the Hospital for Tropical Diseases, of which I am one. No one, I would assure him, would hail with greater pleasure than Dr. LOW and myself the discovery of a real cure for trypanosomiasis. At any rate, I can assure him that we will continue to follow this work with very great interest, but he must remember that we have been following up the few cases for over two years, not for a period of a few months.

Dr. C. F. HARFORD: I do not wish to enter into the technical aspect of this question. It is 31 years since, in Nigeria, I knew of sleeping sickness, and my interest in it has been maintained ever since. I think there is a difference in the cases treated in the country in which the disease has arisen and those which have filtered through into this country and are treated here; they cannot, I think, be put into the same category. In the trypanosomiasis-infected countries there are far greater opportunities of having light thrown on the matter than here, and that may be one cause of the difference in the results of observation.

I hope this matter will be taken up by the Colonial Office; I am sure it must have struck all of us who have heard this remarkably interesting story that it is a matter which should be probed to the bottom.

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The PRESIDENT: I think there has been some misapprehension in regard to treatment. Dr. MANSON-BAHR has spoken about kharsivan, antimony and other drugs, and so has Dr. LOW. The kharsivan is only an activator of the ferments which are in the body. The injection of drugs into the circulation or into the spinal cord is not the only or even the chief basis as I understand of this new treatment of trypanosomiasis. It is only a part. The treatment is to make use of the trypanolytic properties of a serum which has in a high degree acquired those properties by several combined processes, such as the natural increase of power of enzymes or antibodies secreted in the blood and tissues when the body is invaded, the further increase of enzymes or antitoxins on the multiplication and death of trypanosomes in the blood while in the body, and the still further addition of enzymes to the serum on the death of the trypanosomes after the blood containing them has been withdrawn from the body and kept in a vessel.

Dr. MARSHALL: After so much has been said in the discussion, it is difficult for me to take up the points which have been raised.

I am grateful to Professor HALLIBURTON for the way in which he has cleared up the point in regard to the choroid plexus—one which we did not understand before.

Dr. MANSON-BAHR and Dr. LOW quote cases of malaria and syphilis as good parallels. In some cases quinine and salvarsan respectively have no effect on these diseases; but surely that is no reason why quinine and salvarsan should not be tried in ordinary routine cases. Dr. LOW spoke of the results in natives. The European has a higher resistance to the trypanosome than has the native, and if one has cases of natives going two years without treatment and remaining well, it is a sign that at any rate they have improved, even if they have not been cured. With regard to Mr. Rubie, and the possibility of his not having been cured, he does not look very ill at the present moment.

I am very grateful to his Excellency, Sir HUGH CLIFFORD, for what he said in regard to administrative support, and on behalf of Dr. VASSALLO and myself, I wish to say we shall welcome any further tests in order to carry these results to absolute completion, so as to decide, one way or the other, whether or not there is anything in the treatment

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SOME POINTS CONCERNING TRACHOMA.

BY

LIEUT.-COLONEL H. KIRKPATRICK, I.M.S.

It is hardly necessary to lay stress upon the importance of trachoma when addressing medical men who are practising or who are about to practise in the tropics. As you are well aware, the disease is one of the great causes of blindness and loss of efficiency in the Near and Far East.

Trachoma is associated with bad housing conditions, dirt, and general insanitary surroundings. When dust and glare, which lower the resistance of the conjunctiva to infection, are added to these conditions, then a state of affairs highly favourable to the spread of the disease is produced. The zenanas of the poorer class Mohammedans form never-failing reservoirs of infection from which the disease can spread, first to other members of the family, and then to those outside the family circle. The henna pot, which contains a pigment for beautifying the eyes, and which is shared, in a communistic fashion, by the ladies and children of the family, is a fruitful source of infection.

Flies may occasionally be responsible for the spread of the disease, but many authorities now consider them to be an unimportant factor in the dissemination of infection.

Trachoma is common in the Eastern parts of Europe where insanitary conditions prevail, but, fortunately, it is now rare in England, though this country, in common with other parts of Western Europe, was flooded by a wave of infection in the beginning of last century on the return of the soldiers who fought in the Napoleonic wars. The backwash of this wave is yet apparent in Ireland, the less progressive districts of which country are still infected. In England the disease is now chiefly seen amongst immigrants from Eastern Europe.

Trachoma (τραχυς = rough) is a specific and infectious inflammation of the conjunctiva, of a chronic type, and is characterised by the

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formation in the membrane of follicles and papillæ which end in cicatrisation. The disease is frequently called granular ophthalmia, an unfortunate term, since it lays emphasis upon the occurrence of follicles and papillæ which are merely the reaction of the conjunctiva to any form of chronic irritation, and are in no way pathognomonic of trachoma.

Before proceeding further, I propose to recall to your memory certain facts concerning the structure of the conjunctiva. The conjunctival membrane, which has a close relationship to the skin, is continuous with the skin at the margins of the lids. It sweeps backwards over the tarsal plates, to which it is intimately attached, and, forming a very lax fold, or fornix, above the convex border of the tarsus, passes on to the anterior surface of the globe and gives a loose covering to the front portion of the scleral tunic. The conjunctiva is represented on the surface of the cornea by the corneal epithelium.

There are thus three separate parts of the conjunctival membrane: (1) The tarsal or palpebral; (2) the fornical or transitional; (3) the bulbar. The tarsal and fornical parts of the conjunctiva are covered by laminated cylindrical epithelium and there is a considerable development of low papillæ in the former. As these papillæ are covered by epithelium, they do not become evident on the surface until inflammation causes them to hypertrophy. At the junction of the tarsal and fornical portions of the conjunctiva these papillæ become higher and the epithelium dips between them, so that the discrete structures may give a velvety appearance to the membrane. This appearance is very marked in the presence of inflammation, but it may also be found in some cases of simple hyperæmia caused by an error of refraction. A delicate reticulum of adenoid tissue appears in the deeper layer of the conjunctiva at an early age, and is best marked in the transitional portion of the membrane. This tissue may show follicular aggregations even in the healthy membrane, but such formations are much more evident if any chronic source of irritation is present.

Pathology of trachoma.—A section of a trachomatous conjunctiva shows a general round-cell infiltration of its deeper layer with a varying degree of vascular engorgement. Pseudo-papillæ, formed by folding of the swollen membrane, are found, especially in the transitional part of the conjunctiva; tubular depressions, lined by epithelium, are also seen, and cysts may be caused by the occlusion of their orifices or of the

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mouths of the interpapillary crevices and by the subsequent accumulation of epithelial debris. The epithelium is thickened and is infiltrated by leucocytes, and actual downgrowths of epithelial cells may occur into follicles which have ruptured.

The follicle is the characteristic and striking histological feature of trachoma; it is composed of a central group of large connective tissue cells having rounded or oval, pale nuclei, and of a peripheral group of small and deeply staining lymphocytes; the centre of the follicle appears to be paler than the periphery for this reason. The central cells may be mononuclear leucocytes, or may be connective tissue cells, and often appear to be epithelioid, and their cytoplasm may contain cell inclusions of various kinds. Other forms of leucocytes may also be found in this zone. The peripheral cells are enclosed in a stroma which is continuous with the connective tissue of the surrounding conjunctiva, but it is a disputed point as to how far this stroma extends into the interior of the follicle. The follicles are surrounded by a vascular network from which capillaries pass inwards, but these do not appear to reach the centre of the follicle. The existence of a capsule, in the early stages of the follicle, has been disputed, but a capsule undoubtedly forms in the later stages and takes its part in bringing about cicatrisation. It has been stated that the capsule begins to form on the deeper surface of the follicle, and that it is best developed in that part. The follicle ends in cicatrisation, which is generally preceded by a degeneration and softening of its contents. The softened contents may, occasionally, be discharged on the surface, the covering epithelium having become thinned and broken through; but an ingrowth of fibrous tissue, followed by absorption, is the more common termination.

Infiltration of the tarsus by round cells, which is best seen along the course of the vessels, is found at an early stage, and this is followed by distortion and, in some cases, by a partial atrophy of the tarsal plate. The tarsal glands undergo degenerative changes, and the formation of cystic spaces in them is fairly common.

Pannus is seen as a cellular infiltration of the superficial layers of the cornea, accompanied by a rich formation of young blood-vessels. The infiltration, in the early stages, is usually quite superficial and lies between the epithelium and Bowman's membrane, but in the later stages the *substantia propria* is involved, and Bowman's membrane may

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be destroyed in advanced cases. Nodular aggregations of lymphocytes may be found which resemble the young follicles seen in the conjunctiva, but typical follicles rarely occur except upon the limbus.

No specific organism has yet been discovered to be the cause of trachoma. PROWAZEK and HALBERSTAEDTER have described groups of corpuscles in the epithelial cells which they considered might be the cause of the disease. Similar appearances have, however, been found in other conditions, and even in the apparently normal conjunctiva. HEYMAN has found identical forms in cases of blenorrhœa, and GREEF states that "the conclusion that everything, in which trachoma bodies can be found, is trachoma, can no longer be sustained."

Symptoms and Signs of Trachoma.—The disease may be divided into three stages:—(1) An early or florid stage, which is chiefly characterised by the formation of follicles and papillæ; (2) a middle or early cicatricial stage, in which traces of fibrous tissue can be distinguished by the unaided eye as it forms in and amongst the trachoma follicles; (3) a late stage, in which the tarsus and cornea show marked changes, due to the disease, and in which the cicatricial changes in the conjunctiva are very advanced. It is scarcely necessary to mention that no sharp line can be drawn between these different stages, which gradually merge into one another; the forms of the disease may also be considerably modified by the occurrence of secondary infections.

First Stage.—The appearance of small papillary elevations, accompanied by a few irregularly placed follicles, is usually the earliest sign of trachoma. These signs are often said to make their first appearance in the lower fornix, but, at a very early stage, they are best seen at the convex upper edge of the superior tarsus, and, indeed, this portion of the conjunctiva bears the brunt of the attack during the greater part of the disease. Follicles increase in number and are seen on the tarsal conjunctiva as small, raised, yellowish, and irregularly placed spots. Their growth in this region is limited by the intimate connection that exists between the membrane and the tarsus, but they reach a much greater size in the transitional conjunctiva, where their growth is less restrained, and here they are of varying sizes and are pale and translucent, resembling sago grains or frog-spawn. Considerable swelling of the transitional conjunctiva accompanies the formation of the follicles, and small folds and creases of this part of the membrane are formed and lead

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to the appearance of coarse papillæ. The papillæ, which normally exist in the tarsal conjunctiva, undergo hypertrophy as a result of the irritation, and, by doing so, often hide the follicles. The proportion of follicles to papillæ varies in different cases and sometimes one predominates and sometimes the other, so that a "papillary" form and a "follicular" form of trachoma have been described; it must be remembered, however, that the follicle is the essential feature of this stage, and that, though it may not be immediately apparent, it is never absent. The occurrence of a secondary infection will favour a papillary outgrowth and so cause a papillary type of the disease.

It is necessary to evert the fornix thoroughly in order to examine the transitional conjunctiva properly; this may be effected by many different means, of which eversion of the lid by a small button-hook is very effective, but *by far the most satisfactory method is to balloon the conjunctiva by injecting a syringeful of normal saline beneath it*; any changes in the membrane can then be accurately observed as it lies stretched and fully exposed. This method has the further advantage, as will be pointed out later, that it allows remedies to be thoroughly applied to the diseased area and is otherwise beneficial. The occurrence of follicles in the fornix is usually limited by a fairly regular line that lies just anterior to the junction between the transitional and the bulbar conjunctiva.

Ptosis can be nearly always observed at this period, even in the early stages; this drooping of the lid is most probably due to a loss of function of the levator palpebræ owing to its insertion having become involved in the inflammation. *Pannus*, or vascularisation of the cornea accompanied by some opacity, due to subepithelial infiltration, may or may not be present in this stage, but, if the cornea is carefully examined, it will usually be possible to detect a slight swelling of the upper part of the limbus associated with the presence of some fine vascular twigs, which branch on to the neighbouring corneal tissue.

Middle Stage.—All the signs become more accentuated in the middle stage of the disease. Ptosis is obvious and pannus becomes more marked, and often involves the whole of the upper half of the cornea. If the transitional conjunctiva is ballooned by an injection, delicate lines of fibrous tissue can be seen running through it, so that it appears as if a fine grey net had been pressed into the membrane and had been

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incorporated with it. A line of fibrous tissue may also be observed to run horizontally about 3 mm. above the free edge of the lid in the *sulcus subtarsalis*, at the point where the *rami perforantes* pass from the inferior tarsal arch of vessels to the conjunctiva through the tarsal plate. This line becomes broader and deeper, and is known as *Arlt's line*. The internal margin of the lid, which normally has a sharp and well-defined edge, becomes rounded, and a soft granulation tissue often forms upon it. When the follicle formation is in excess, the follicles on the tarsus sometimes become aggregated into an almost homogeneous, firm, gelatinous mass, which is of pale yellow colour; a considerable thickening of the lid may be produced in this way; the surface epithelium becomes thickened at the same time, and the whole syndrome forms the condition which is known as *Stellwag's gelatinous trachoma*. Cysts may also form in the conjunctiva as the result of the retention of cell products in the crevices between the papillæ and of the accumulation of glandular secretion. Signs of the previous existence of trachoma can often be observed, even if the disease has been cured in the early part of its middle stage. The tarsal conjunctiva will, in that case, be seen to have a faint lilac tinge, which is quite characteristic, and some contraction of the fornix will usually be apparent. In the later part of the middle stage the shrinking of the fornix is much more striking, and it may be possible to recognise definite strands of fibrous tissue in the conjunctiva.

Third Stage.—The changes in the third stage are due to shrinking of the new-formed fibrous tissue and to degenerative changes in the tarsal plate. The tarsus becomes rather sharply bent, so that an angle, whose apex looks forwards, is formed in the lower part of the plate. The tarsus is also reduced in size, and may, in some cases, be merely represented by a small rod-shaped mass of tissue, which contains fat, cheesy matter, and fibrous tissue. These alterations are due both to the degeneration of the tarsal plate and to the traction of the shrinking conjunctiva upon its deep surface. The bending of the tarsus leads to an entropion, so that the lashes turn inwards, and a trichiasis, or rubbing of the cilia on the cornea, adds to the corneal irritation and pannus. Shrinking of the conjunctiva leads to a diminution in size of the transitional fold, and much of the anterior layer of this fold is drawn down to form a covering for the upper part of the tarsal area of the lid. The conjunctival surface of the tarsus shows white branching strands of scar

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tissue, and between these strands may be seen small spots of granulating tissue which represent the remains of hypertrophied papillæ. The lid margin has a sinuous outline, described by HERBERT, the portion near the outer canthus being at a lower level than the central and inner parts. The size of the palpebral aperture is also often lessened. Pannus is very marked in this stage and corneal ulceration may occur; the corneal troubles are much aggravated by any trichiasis which may be present. In very severe cases, where the cicatricial changes and shrinking of the fornix have led to an excessive atrophy of the conjunctiva, changes occur in the surface epithelium, the uppermost layer of cells undergoing keratosis with a general deposition of keratohyalin, so that the membrane becomes epidermoid in character. Under such circumstances the eye becomes dry and no tears adhere to the surface of the diseased conjunctiva. This condition of *xerophthalmia* is not limited to the conjunctiva, but affects the surface of the cornea, and, by causing its opacity, leads to blindness.

Diagnosis of Trachoma.—It must be admitted that the diagnosis of trachoma, before the cicatricial stages have set in, is a problem of considerable difficulty, and even a surgeon of considerable experience will frequently find himself unable to decide whether a conjunctival disease is truly trachomatous or not. These difficulties are not so pronounced, of course, in the later stages, when cicatricial changes have occurred. The reason is that follicle and papillary formation are the natural reaction of the conjunctival membrane to many different varieties of irritation, infective and otherwise. The follicle formation which results from the excessive use of atropine in susceptible persons is an example of one of these, and I have known cases of irritation from this cause to be diagnosed as trachoma. This difficulty of diagnosis must necessarily continue as long as we are unable to identify the causative organism of the disease. Three signs are of value as an aid to diagnosis in the early stages of the disease; these are (1) the presence of ptosis, (2) the early occurrence of pannus in the upper part of the cornea, (3) the situation of the trachoma follicles. Ptosis, of some degree, is seldom or never absent, and the patient acquires a peculiar sleepy appearance owing to the drooping of the upper lid. Pannus appears in that part of the cornea which is in constant contact with the rough and infected membrane, and is first seen at the upper part of the limbus, but the

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vascularised area of infiltration soon encroaches upon the upper part of the cornea; this area is generally limited by a fairly well-defined and regular horizontal margin below, which, in the early stages of the disease, does not ordinarily reach lower than the level of the upper border of the undilated pupil. Vascularisation, which commences in parts of the cornea other than its upper half, is unlikely to be due to trachoma.

The follicles, in trachoma, are best marked along the upper, or convex margin, of the superior tarsus, and from here they spread upwards to the fold of the fornix, and downwards on to the conjunctiva which covers the tarsus. They may be seen to a much less extent in the lower fornix and the conjunctiva of the lower lid, and, in some cases, they may even be found upon the plica semilunaris. The follicles are of varying size and are rather irregularly placed, whilst some may be masked by the development of papillæ.

The conditions which are most difficult to distinguish from the earlier stages of trachoma are (1) Follicular conjunctivitis, (2) spring catarrh, (3) a chronic form of purulent ophthalmia.

Follicular conjunctivitis is the disease that presents the greatest difficulty in diagnosis, since it also is characterised by the appearance of follicles, which, however, differ from those of trachoma in that they do not undergo cicatricial changes. Follicular conjunctivitis is not usually accompanied by ptosis, and, unless the conjunctiva is irritated by caustic applications or by secondary infections, shows little tendency to papillary formation or to pannus. The follicles are regular in size and in shape, and are as marked in the conjunctiva of the lower lid as in that of the upper; indeed they may be often rather more so. Follicular conjunctivitis is usually a condition of childhood, and is often associated with an excessive development of adenoid tissue in the naso-pharynx, in the cervical lymphatics, and in other parts of the body. A hyperæmia, induced by an uncorrected error of refraction, or by a mild secondary infection, will sometimes increase the follicular growth and cause the formation of papillæ and so render the diagnosis especially difficult. In cases of difficulty it is advisable to use mild lotions, to correct the refraction, and to watch the course of the case; if the disease shows no signs of progress, it is not true trachoma.

Spring catarrh is characterised by a papillary formation in the conjunctiva of the tarsus and in the transitional conjunctiva. The

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Fig. I.

tion under low magnification showing tarsus, sal conjunctiva, and transitional conjunctiva he early part of the second stage of trachoma. chomatous follicles and interfollicular de- ssions are seen in the transitional conjunctiva.

To accompany Col. Kirkpatrick's Paper (Face p. 42).

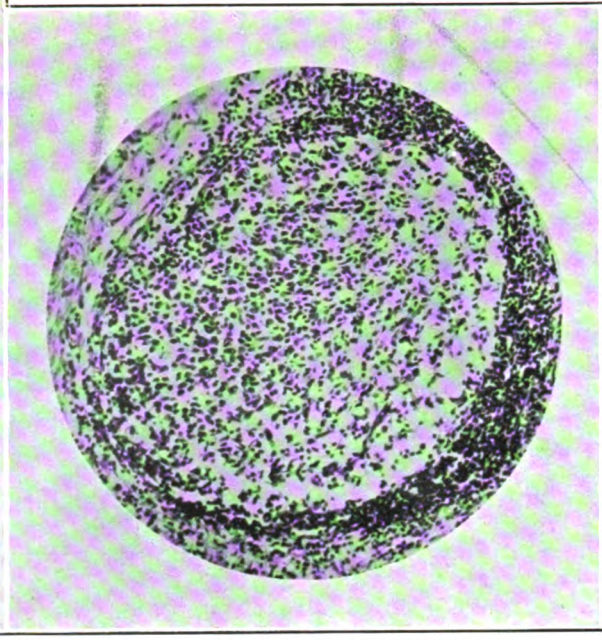

Fig. II.

Trachoma follicle showing peripheral zone of small, deeply-staining cells and central area of large cells. Young blood-vessels seen in periphery, and early capsule formation.

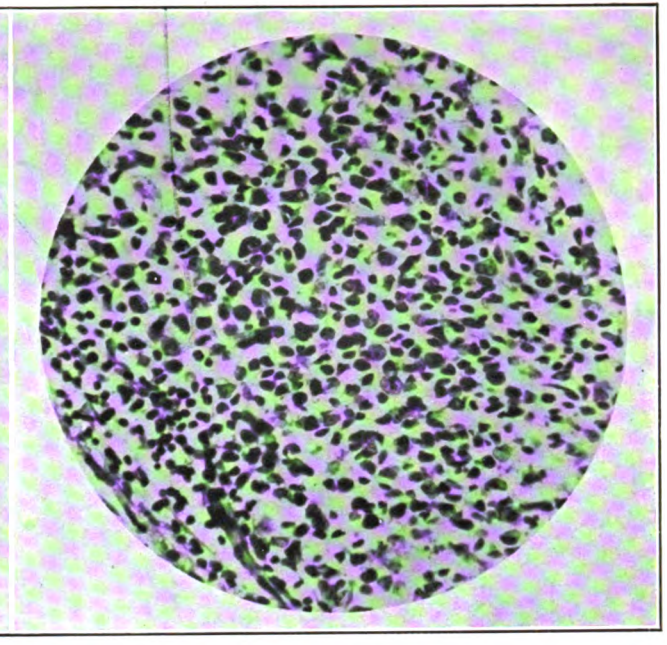

Fig. III.

Trachoma follicle, central zone. Note variety of cells.

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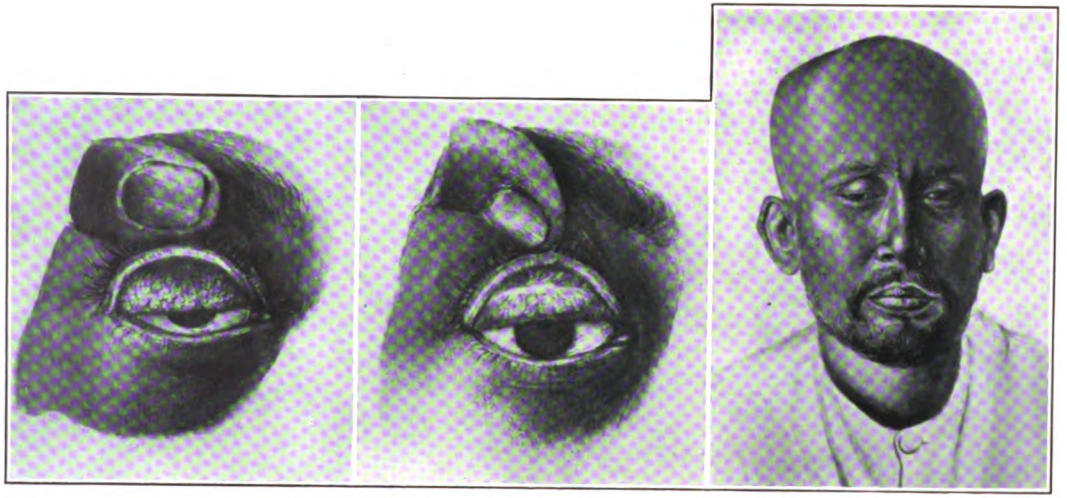

Fig. IV.
Later part of First Stage.

Fig. V.
Later part of Second Stage. Ault's line is well marked. Note the presence of soft granulation tissue on margin of the lid.

Fig. VI.
Third Stage.—Ptosis and the bulging forward of the tarsus seen in right eye. Herbert's sinuous lid margin seen in left eye.

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papillæ are discrete and firm, with flattened tops, and, unless inflamed, are pale and pink with a peculiar bluish tinge. The colour is sometimes compared to that of ham. The papillæ are thus quite different in appearance to the softer and readily bleeding papillæ found in trachoma. The conjunctival discharge contains a considerable number of eosinophile leucocytes, and the disease is usually accompanied by an eosinophilia. An absence of follicle formation is a further point of differentiation between the diseases. A spring catarrh, associated with a secondary infection, may present considerable difficulties in diagnosis from trachoma; in such cases a careful examination should be made of the discharge for the presence of eosinophiles. Little difficulty will be experienced in forming a diagnosis if the characteristic corneal changes of spring catarrh are present. Trachoma and spring catarrh are sometimes combined, and such cases will be difficult to diagnose and to treat correctly.

High papillæ, which are soft and bleed readily, and which are accompanied by some irregular follicles, are sometimes found in cases of *purulent ophthalmia*, the acute stage of which has passed off. The papillæ, in cases of this sort, are usually larger than those found in trachoma and a thin, greenish-yellow discharge may be seen to trickle from the spaces between them. An examination of the discharge will show the presence of the causative organism, which is usually the gonococcus. Pannus is absent in these cases, but there may be a vascularisation of the corneal edges, which is not confined to the upper part of the cornea. An inflammation of this character will yield to suitable treatment more readily than trachoma will; the disappearance of the papillæ, after a short course of treatment by eyebaths of magnesium sulphate lotion, aided by the use of copper sulphate, will show the nature of the disease. It should be remembered that trachoma is sometimes complicated by the onset of an attack of gonorrheal ophthalmia; this is said to occur very frequently in Egypt.

The hyperæmia and other conjunctival changes, produced by *an uncorrected error of refraction, is the condition which is most often confused with trachoma by those who are unfamiliar with eye diseases.* Such a refraction error frequently causes a hyperæmia of the conjunctiva and the eyelids, and an increased glandular activity follows the excessive supply of nutriment. This is shown, not only by watering of the eye, but also by the collection of secretion along the edges of the lids, and,

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occasionally by the formation of so-called Meibomian cysts or chalazia. The redundant secretion affords a medium in which micro-organisms can grow and multiply, and its presence lowers the resistance of the conjunctiva to infection, so that inflammations of the membrane are common in such cases. Small papillæ and follicles may be found in the transitional conjunctiva, and, as previously stated, if these are irritated by the application of caustic drugs, the hyperæmia is increased and a condition closely resembling true trachoma may be produced. The result of treatment will usually show the nature of the case. Patients who have a seborrhœic tendency and who suffer from an error of refraction, appear to be specially liable to this condition, and the use of resorcin (in 1 per cent. eyedrops), in addition to the correction of the refraction error, is often valuable in such cases.

The Treatment of Trachoma may be considered with reference to the stage of the disease and with reference to whether it should be medicinal or operative.

Treatment should be directed towards promoting the disappearance of the follicles without, at the same time, causing the formation of an excessive amount of fibrous tissue. Nature will, in course of time, effect the first object, but she does so by a cicatrisation that leads to very undesirable complications and sequelæ.

The first essential is that the patient should be placed under good sanitary and hygienic conditions, so that he may be kept in as good a state of general health as possible, and that he may cease to be a source of danger to others. Good food, fresh air, general cleanliness, and suitable tonics fulfil the first requirement. The provision of separate basins and towels for the patient, the observance of proper precautions regarding the cleansing of the eyes by attendants, and the right disposal of cloths soiled by conjunctival discharge, will, under ordinary circumstances, satisfy the second. Should the disease appear amongst members of a community, such as those living in schools or barracks, infected persons should be segregated and placed in special companies or taught in separate classes. The mistake made at the beginning of last century, when infected soldiers were discharged to their homes, and thus allowed to disseminate the disease broadcast, should be avoided, and these patients should not be allowed to return to their families until they are free from infection.

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For the treatment of the first stage of the disease, nitrate of silver solution and copper sulphate, or blue-stone, are probably the two drugs which are most in favour as medicinal applications. Silver nitrate is used in various strengths, from 5 grains to the ounce of distilled water to 60 grains to the ounce, but a solution of 10 grains to the ounce will usually be found to be the most satisfactory; the stronger solutions are too irritant and are likely to cause too great a formation of fibrous tissue, whilst the weaker ones are insufficient to cause a proper reaction and to promote absorption. A mop, made by twisting a little cotton-wool on to the end of a small stick (for example a match), is dipped into the solution and then applied to the affected area, the excess being washed off by a stream of saline solution after a pause of about twenty seconds. This may be applied daily or three times in a week according to circumstances. The application is followed by the formation on the surface of the conjunctiva of a bluish-grey film, composed of necrotic epithelium; this is shed in the course of a few hours. Patients occasionally complain that they notice haloes around lights after treatment by silver nitrate; this symptom arises from the effect of the drug upon the corneal epithelium, changes in which cause a dispersion of light. The appearance is only a temporary one, but patients may be alarmed by its occurrence, since a similar symptom is common in glaucoma.

Sulphate of copper should be applied in the form of a stick, or of a crystal which has a perfectly smooth surface. The retrotarsal fold is thoroughly exposed and the crystal, or stick, is lightly rubbed over any prominences which are seen on the surface of the conjunctiva; the conjunctival sac is then thoroughly irrigated with saline solution. The use of copper sulphate is specially indicated in cases where papillary formation is a marked feature. Strong solutions of perchloride of mercury (1 in 100 or 1 in 500) have been recommended by some surgeons; these are rubbed into the affected area with a mop, the pressure of which at the same time forces out the contents of the follicles. Some advocate the application of boracic acid powder in the same manner, and this has the advantage that it causes little irritation. HERBERT recommends intensive irrigation with 1 in 3000 perchloride of mercury solution. Whatever drug be favoured, it is essential to apply it to that part of the conjunctiva which is diseased, and the fornix must be thoroughly everted for this purpose. *A subconjunctival injection of fluid, which balloons out the*

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fornix, will be found to be a most efficient method of ensuring a complete exposure of the membrane. The upper lid is everted and the point of the needle is entered at the outer angle of the upper margin of the tarsus, and a syringeful of normal saline solution is injected. If 4 minims of a 4 per cent. solution of cocaine, or similar substance, and 4 minims of adrenalin are added to the saline, any subsequent manipulation of the conjunctiva will be rendered painless. A subconjunctival injection has further great advantages, since (1) it stretches the membrane and so tends to lessen the contraction caused by cicatrisation, and (2) it promotes the absorption of inflammatory products by a lymphagogic action. Puncture of the follicles by a needle or by the point of a galvano-cautery has been recommended by some surgeons of experience, but the expression of their contents is probably the most efficient method of dealing with the follicles at this stage. Expression is carried out by means of a forceps, between the blades of which the diseased membrane is squeezed. The blades of some of such forceps are furnished with rollers in order to facilitate expression of the follicles. A preliminary ballooning of the fornix by the subconjunctival injection of an anæsthetic fluid is advantageous in all these methods of treatment.

The application of carbon-dioxide snow and exposure to X-rays have been recommended in the treatment of trachoma, but these measures have not proved entirely satisfactory in my experience.

It must be borne in mind that trachoma is an obstinate disease, and that, even if detected in the first stage, it will require treatment for some months, if all danger of relapse is to be avoided. If the disease fails to respond to treatment, or if the patient is unable to afford the time necessary for a thorough cure, it may be advisable to undertake a more radical measure and to excise the transitional portion of the conjunctiva.

Such an excision is a simple proceeding; the lid is everted and the fornix ballooned; a slightly curved, horizontal incision is made beneath the follicle-bearing area, and its extremities are then joined by a similar incision passing along the upper margin of the tarsus. These incisions should be quite superficial and should merely divide the conjunctiva. The elliptical area, thus isolated, is then easily lifted from the subjacent tissue by the knife or scissors; the cut edges of the membrane may be slightly undermined and then united by three or four sutures, but this is not really essential, as healing takes place rapidly. This little operation is followed by very little trouble or discomfort.

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A trachomatous conjunctiva, owing to its impaired power of resistance, is very subject to the occurrence of secondary infections, and these should be carefully watched for and appropriately treated when recognised. Inflammation due to the Morax-Axenfeld bacillus is one of the most common of such infections, and this will usually readily yield to treatment by sulphate of zinc drops. Koch-Weeks infections are best treated by silver nitrate, and eyebaths of a saturated solution of magnesium sulphate will be found useful in the case of infection by the gonococcus. The latter form of treatment is also of great value in inflammations due to streptococcal infection.

The treatment of the second stage of the disease is conducted on the same lines as that of the first stage, but, owing to the presence of the newly-formed fibrous tissue, medicinal treatment is unlikely to be as effective in this case, and operative measures are more likely to be required. Copper sulphate is usually more efficacious than silver nitrate at this stage, but the course of treatment will probably be much shortened if more radical treatment is adopted. Colonel HENRY SMITH recommends the employment of subconjunctival injections of cyanide of mercury, and ABADIE has recently reported excellent results from deep and multiple punctures of the tissues at the upper margin of the tarsus by a galvano-cautery; the punctures are repeated until a regular trough is formed in the tissues. It will be found, however, that the *combined excision of the fornix and tarsus*, as described by HEISRATH, will probably prove the most satisfactory form of operation. This operation can be very easily carried out if attention is paid to the following steps:—

After ballooning the fornix by the injection of a local anæsthetic combined with adrenalin, a suture is passed through the thickness of the lid at the central point of its margin; this suture serves as a landmark, and assists to control the lid. The upper lid is everted, and the retro-tarsal fold is seized by a fixation forceps and thoroughly exposed; a fixation forceps, provided with a catch, is applied to the outer part of the fold (the inner part in the case of the left eye) and allowed to hang free, in such a manner that full eversion is obtained (Fig. VII). The inner part of the fold (or the outer in the case of the left eye) is next grasped by a forceps held in the left hand, and the tissue is stretched between the two pairs of forceps. A horizontal incision is made through the conjunctiva, just below the follicle-bearing area, along the whole length of the lid

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(Fig. VII, 3). A sharp knife is required for this purpose, and care must be exercised to keep the plane of the blade at right angles to the surface of the membrane. The conjunctiva, which forms the lower edge of the wound, is then thrown down towards the globe, so as to form a loose fringe of membrane (Fig. VIII, 5). The fornical conjunctiva is next peeled upwards off the underlying tissues as far as its attachment to the tarsus. This dissection is made more by pushing movements of the blade of the knife than by actual cutting, and it is usually only necessary to divide a few strands of tissue by the knife edge. The extremities of the wound are next joined by a second incision which passes close to the border of the lid (Fig. VII, 4); the centre of this incision is deepened until the tissues which underly the tarsus are exposed (Fig. VIII); the incision in the tarsus is then completed by seizing the lower margin of the wound, already made, in a forceps, and by extending the cut laterally through the entire thickness of the tarsal plate. The tarsal plate is next dissected off the tissues beneath as far as its upper margin, so that it only remains in position along a narrow horizontal line; the tarsus and the attached apron of transitional conjunctiva are now held up by a forceps, between the blades of which both structures are grasped, and the remaining attachment is divided by a few strokes of the knife or by a few snips of the scissors. A fairly extensive, elliptical, bare area is left by the removal of the tissue (Fig. IX, 4). The fringe of conjunctiva, which remains continuous with the bulbar part of the membrane, is raised, and a suture is passed through its margin, at a point which would be intersected by a perpendicular line let fall on to the extremity of the horizontal diameter of the cornea; the needle is again passed through the cut edge of the tarsus at a point 6½ mm. to one side of the suture marking the central point of the lid margin (Fig. IX, 5). A second suture is passed at corresponding points at the other side of the wound, and, when these are tied, the operation is completed. It is very important not to injure the deeper tissues when stripping off the conjunctiva and the tarsus, and to place correctly the sutures, which unite the edges of the wound. Bleeding may be troublesome occasionally, but is seldom likely to be so if adrenalin is injected with the local anæsthetic. The operation is followed by some reaction, which lasts for about thirty-six hours and then passes off quickly. The sutures may be removed on the third or fourth day, and painting with silver nitrate may be commenced at the end of the first week.

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The treatment of the third stage of trachoma is chiefly concerned in the treatment of complications caused by the contraction of the fibrous tissue and by the distortion of the tarsus. The depth of the fornix is greatly diminished, partly because the membrane is shrunken, and partly because it is drawn down over the remains of the tarsus by the contracting fibrous tissue in that region, and there will therefore be less difficulty in applying drugs to its surface. Small, raised, granulating spots, which bleed easily, may be seen amongst the fibrous tissue, and the application of copper sulphate to these is beneficial. The chief indication is, however, to treat any complication, such as trichiasis, that may be present, and to recognise and to remedy any of the secondary infections that are so commonly found at this stage of the disease. The operation of excision of both fornix and tarsus is not advisable, since so little of the transitional conjunctiva remains, but much benefit may be derived from the excision of the tarsus, the diseased tissue of which often appears to serve as a reservoir of infection.

Pannus is best treated by attention to the disease of the conjunctiva, and an improvement of the corneal condition will quickly follow an improvement in the state of that membrane. A peritomy will sometimes be of advantage in helping to clear up a very vascular pannus, and a subconjunctival injection of cyanide of mercury will often hasten the disappearance of opacities. The slightly facetted, chronic ulceration, that so often persists after the conjunctiva has been practically cured, may be much benefited by the use of Scarlet R applied once or twice daily as a 2 per cent. solution in olive oil. I have also found this application to be very valuable in early cases of xerophthalmia. Dionine is often of use in promoting the absorption of opacities due to pannus. A very marked improvement in pannus nearly always follows the combined excision of tarsus and fornix. The state of the lachrymal apparatus should be carefully investigated and watched, as an infection of the canaliculi may occasionally cause a persistent corneal irritation. Jequirity is highly recommended by some surgeons for the treatment of pannus. The instillation of the drug induces a severe inflammation of the conjunctiva, and this often leads to the shrinking of the vessels which have invaded the cornea. The conjunctiva is painted every day with an infusion made from crushed jequirity seeds; the infusion is used in a 5 per cent. solution. The application is stopped when a sufficient reaction has been produced.

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It is advisable for the patient to use eyedrops of ½ per cent. solution of copper sulphate or of a 7 per cent. solution of tannic acid in glycerine for some weeks after discharge from hospital; a yellow oxide of mercury ointment should be applied at bedtime.

In conclusion, I would like to suggest that, even in apparently typical cases of trachoma, we may not be dealing with a single disease entity, and that the causes of the conjunctival changes, characteristic of trachoma, may widely vary in different cases.

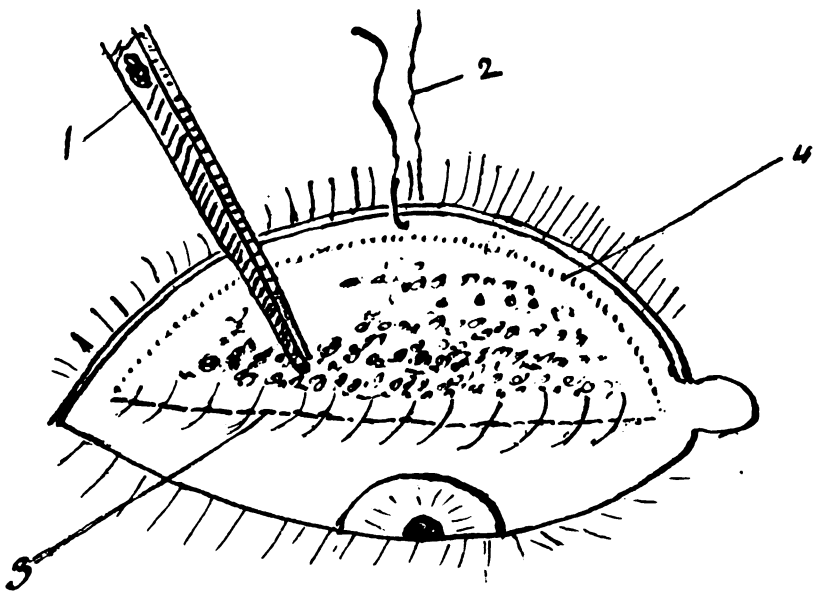

Fig. VII.

1. Fixation forceps, with catch, holding retro-tarsal fold everted.
2. Suture marking central point of lid
3. First incision, made through the transitional conjunctiva, just below the follicle-bearing area
4. Incision through tarsal conjunctiva; this is made to divide the tarsal plate in the middle portion.

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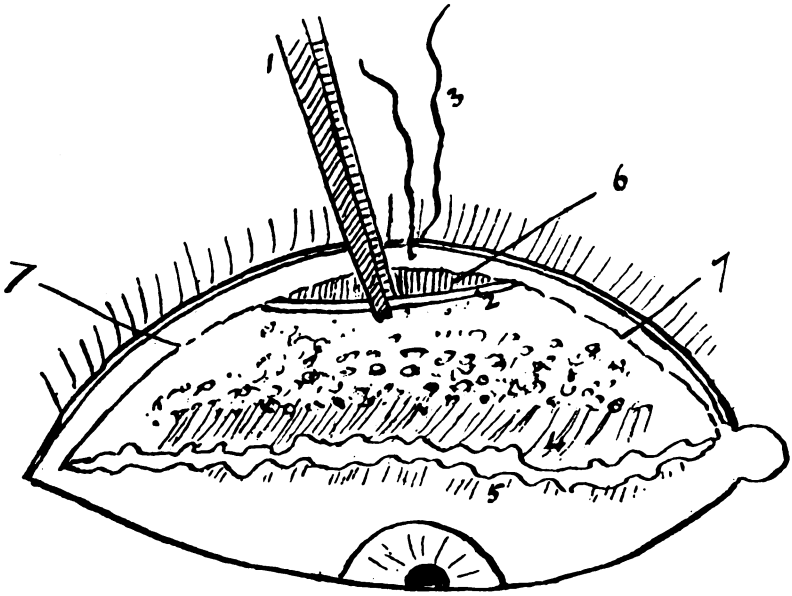

Fig. VIII.

1. Forceps gripping the edge of the divided tarsal plate.
2. Cut edge of tarsal plate.
3. Suture marking point in lid which lies over the centre of the cornea.
4. Detached fringe of transitional conjunctiva.
5. Detached fringe of bulbar conjunctiva.
6. Tissues, which underly the tarsal plate, exposed by throwing the divided portion of the tarsus downwards.
7. The incision through the tarsal conjunctiva.

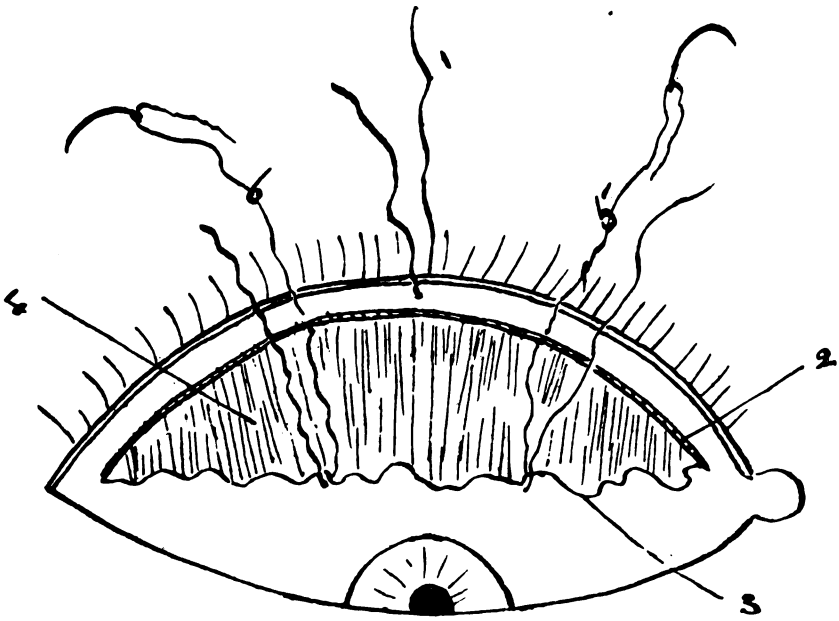

Fig. IX.

1. Suture marking the point in the lid which lies over the centre of the cornea.
2. The cut edge of the tarsal plate.
3. The cut edge of the fringe of bulbar conjunctiva.
4. Deep tissues, exposed by the removal of the tarsus and transitional conjunctiva.

5 and 6. The sutures, by which the fringe of bulbar conjunctiva is united to the cut edge of the tarsus. These are passed through the edge of the bulbar conjunctiva just to the outer side of a point which would be intersected by a perpendicular line let fall on to the extremity of the horizontal diameter of the cornea. They are passed through the cut edge of the tarsus at a point 6½mm. to one side of the suture No. 1.

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DISCUSSION.

Colonel R. H. ELLIOT: Colonel KIRKPATRICK said that he had nothing new to tell us. If that is the case, he has certainly put before us a number of old things in a new way. One of the most important parts of his paper is his reference to the large number of conditions which, certainly in my time, we put down to trachoma. Many of the cases which we confidently called trachoma in the old days were not true trachoma. Trachoma is a word for a congeries of diseases, and not a single disease. That is a line along which some important work can be done. I should like to refer to the extraordinary importance of this question of trachoma. I am without the slightest fear of overstating the case when I say that trachoma in India, and I understand also in Persia, Afghanistan, and China as well, is responsible for more blindness than any other two factors put together. Even if you put ophthalmia neonatorum and cataract together, I do not believe that they are responsible for such a big loss of sight in Eastern countries as trachoma alone. But trachoma is, if treated properly from the first, invariably a curable disease. I have had trachoma myself, and have been completely cured of it and can speak of it from the patient's side. If a case of trachoma is treated properly from the start, blindness need never result, and yet trachoma is responsible for more blindness than anything else. It is of great importance that Governments should be awake to their responsibilities in regard to trachoma and other eye diseases. Governments—in India, for instance—have never been anything like awake enough to these diseases and to the possibility of cutting off trachoma at its earliest stages. There are two other points I would like to commend to your attention. One is Colonel KIRKPATRICK's suggestion of the magnesium sulphate treatment. I have had very little opportunity of trying it myself, but I have met with a number of young men who have seen Colonel KIRKPATRICK use it in Madras, and who have come back enthusiastic about its advantages. The other thing I can speak of with more emphasis, viz., the procedure of ballooning the conjunctiva. I believe that this is an extraordinarily valuable manœuvre, both from the point of view of diagnosis and of treatment.

Lieut.-Colonel S. P. JAMES: In view of what Colonel ELLIOT has said about the responsibility of Governments, and the fact that trachoma in its very early stages can be treated and cured successfully, may I ask

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Colonel KIRKPATRICK whether he considers that trachoma should be made compulsorily notifiable in England? That is a question we are frequently asked in the public health service.

Colonel H. KIRKPATRICK: In reply to Colonel JAMES, I am afraid my experience in England is very limited. I used to meet with a good deal of trachoma more years ago than I care to number, when I was a house surgeon in Dublin. I certainly think, however, that it ought to be notifiable. It is an infectious disease and a dangerous one, and consequently it ought to be notifiable.

Lieut.-Colonel JAMES: The difficulty is diagnosis.

Colonel KIRKPATRICK: Typical trachoma certainly should be notifiable.

Colonel ELLIOT: I should like to support Colonel KIRKPATRICK in this matter. It would be much better to have notification, even if a number of the cases turned out not to be trachoma, than to let cases of trachoma be unnotified and go far and wide. After every big war we have had numbers of cases of trachoma in this country, and, in fact, far over Europe.

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TRACHOMA AMONG THE CHINESE IN FRANCE.

BY

CHARLES F. HARFORD, M.A., M.D.

The importance of trachoma from the epidemiological standpoint was clearly shown by experience gained with the Chinese Labour Corps in France, and the object lesson there afforded should lead to new efforts to definitely establish the pathology of the disease; its relation to the chronic forms of trachoma found in England; and the measures which may be adopted to stamp it out, or, at least, to minimise the serious effects which it is producing in many tropical and sub-tropical climates.

My own contact with the Chinese was but brief, but it involved certain vivid impressions which may be worthy of record, and may at least give an opportunity of emphasizing the great difficulties of diagnosis which attach to all diseases which are not identified by distinct pathological proof. I may add that during most of the time in which my observations were made I was not acting as an ophthalmic specialist, as I was doing during the greater part of my military service, but while serving in France in connection with Native Labour Corps, and mainly with those from South Africa.

1. My first touch with the Chinese was in August, 1917, when two Chinese companies arrived at Dannes to take the place of two South African companies, these being taken away by the same train which brought the Chinese. They brought no medical officer with them, and although I was appointed to a camp of South African natives about a mile away across our great ammunition dump, I was called in to help the Chinese until they obtained their own medical officer.

The immediate point which had to be considered was an ophthalmic one. The Chinese had undergone a medical inspection since landing in France, and the two companies which had been sent to Dannes were those who were regarded as suspect from the point of view of trachoma,

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the companies being graded: Company A (as we may call it) being regarded as a "Trachoma Company," and Company B as a "Conjunctivitis Company." This meant that the former were regarded as potentially trachomatous, Company B, on the other hand, being composed of those who had some conjunctival trouble which rendered them suspicious.

Obviously this classification was only one of convenience, and later experience of examining large bodies of Chinese demonstrated the hopelessness of expecting exact diagnoses in such circumstances, especially when the disease itself, in its acuter manifestations at least, has no definite standard which can be regarded as wholly reliable.

As a preliminary step in dealing with these two companies the officers gave instructions for the men who were suffering from the most obvious eye troubles to be paraded for my inspection. Less than fifty were thus brought before me, and of these only a few were of any severity. The worst of these reminded me of bad cases of mucopurulent conjunctivitis seen in England, with the addition of some nodular elevations under the red velvety conjunctiva. Clearly we were faced by the presence of a form of contagious conjunctivitis which needed drastic treatment. This was rigidly carried out in this camp by a vigorous and able Canadian Medical Officer, under the instructions of Sir WILLIAM LISTER, who had visited the camp, and under the supervision of Mr. HINE, who was the ophthalmic specialist of the district. This régime will, I hope, be described by those who were concerned in its inception and execution.

2. The next problem which I was called upon to consider was the possibility of the contamination of the South African natives by their contact with the Chinese. I was able to give my opinion that the system of segregation which was well carried out by South African officers and non-commissioned officers practically excluded any danger on this ground. Certainly I have never heard of any case of trachoma arising in France among South African natives.

3. On my transference to Havre with the South African natives I was once more temporarily associated with the Chinese, when these in turn replaced the companies who were returning to South Africa.

The Chinese sent to Havre were those who had been certified as "clean" companies from the point of view of trachoma, so that the problem was a somewhat different one from that at Dannes. Our D.D.M.S., however, most wisely decided to take no risks, and a

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scrupulous examination of the eyes of the Chinese was decided upon.

I paraded the company assigned to me and determined to pick out every man with red eyes, and detain all such cases for more detailed examination. I then graded these according to their severity, and had the advantage of the opinion of Mr. SICHEL, who had been recently engaged in the investigation of the causes of trachoma in Christmas Island. He gave the very decided opinion that even the few more severe cases were not of trachomatous character. He pointed out that the Chinese, with whom he was familiar, have very loose lids, that they were thus very liable to irritation and consequently accustomed to rub their eyes with dirty hands and towels, so that most of them had some congestion of the conjunctiva. I have no doubt that this was the case, and, on the one hand, it should be recognised that red eyes with these men may mean very little, and on the other hand may predispose them to worse troubles.

4. Later on in my capacity as one of the ophthalmic specialists in the Havre area, I was called upon to make a routine inspection of a large number of Chinese without any cases being found which could be regarded as suspicious.

5. Finally, I should like to bear my testimony to the excellent hospital arrangements made for the Chinese, whether at the central hospital at Noyelles or in that at Havre, in both of which Medical Missionaries from China, or those who had served as such in the past, took an important part in the work.

I am strongly of opinion (*a*) that the term trachoma is too loosely applied to cases of conjunctivitis in sub-tropical climates; (*b*) that the whole subject calls for the earnest attention of ophthalmologists and of all practitioners in these countries; and (*c*) that the governments concerned should make it the subject of searching enquiry.

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AN EARLY PORTUGUESE CONTRIBUTION TO TROPICAL MEDICINE.

BY CARLOS FRANÇA, COLLARES, PORTUGAL.

(Translated and communicated by CLIFFORD DOBELL.)

It is impossible not to recognize the scientific character of the conquests and discoveries of the Portuguese in the fifteenth and sixteenth centuries Nevertheless, in reading treatises on tropical medicine we hardly ever meet with Portuguese names when the early history of the subject is under discussion ; though it would be surprising if the distinguished men among the Portuguese—who showed themselves, especially in Brazil, to be scientific colonists—had left no obvious traces of their activities upon tropical medicine.

Two of the historians of medicine, MAXIMIANO LEMOS and RICARDO JORGE, have noted that Portuguese physicians were the first to mention some of the tropical diseases. For instance, GARCIA DA ORTA (1583) gave a very good account of cholera; ALEIXO D'ABREU (1623) described epidemic gangrenous rectitis (*mal do bicho**), and FERREIRA DA ROSA (1694) yellow fever. Even travellers who were not medical men gave remarkably accurate descriptions of certain diseases, and intuitively recognized the true nature of some of them. The chronicler, JOÃO DE BARROS (1552), noted the symptoms and ætiology of scurvy, from which the ship's company of VASCO DA GAMA suffered; and another chronicler, GASPAR CORREIA, described the cholera epidemic of 1543 with admirable precision; while the Jesuit, GASPAR AFFONSO (1596), gives one of the most complete accounts of *Dermatophilus penetrans.*†

* *Maculo* of the West Coast of Africa; *cheringoza* of Central Africa (CAPELLO and IVENS).

† This description shows that these fleas—contrary to the statements in works on tropical pathology—existed in the sixteenth century not only in Brazil, where they were known by the name of *zungas*, but also in India, where they were called *nigoas*.

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All these facts show that the Portuguese made contributions to our knowledge of the diseases of tropical countries; but not one of them appears to me so interesting as that which forms my justification for publishing the present note.

I have recently found, in a work of the sixteenth century, a very clear statement of the part played by insects in the transmission of Framboesia tropica. In 1587 a Portuguese colonist, named GABRIEL SOARES DE SOUZA, wrote a work containing a very interesting description of Brazil, and in it may be found most complete accounts of the history, geography, and ethnography, and of the fauna and flora of our great American settlement. In this very remarkable work, which contains chapters surpassing those of PISO and MARCGRAF, I have found the following passage: "Digamos logo dos mosquitos, a que chamam nhitinga, e são muito pequenos e de feição de moscas; os quaes não mordem, mas são muito enfadonhos, porque se poem nos olhos, nos narizes, e não deixam dormir de dia no campo, se não faz vento. Estes são amigos das chagas, e chupam-lhe a peçonha que tem; e se vão pôr em qualquer cossadura de pessoa sã, deixam-lhe a peçonha nella, do que se vem muitas pessoas a encher de boubas."*

The literal translation of this is as follows: "We must now refer to those mosquitoes which are called *nhitinga*, and which are very small and of the form of flies [house-flies]; these do not bite, but are very troublesome, because they settle on the eyes and in the nostrils, and will not let one sleep by day in the open, unless there be a wind. They are very fond of sores, and suck the poison which is in them; and if they then go and settle upon any abrasion on a healthy person, they leave the poison in it, and hence many people are seen covered with *boubas*."

It should be noted that *boubas* is still at the present day a popular name for framboesia in Brazil; whilst *buba* is the Paraguayan designation for cutaneous leishmaniasis.† In the Congo, framboesia is known

* GABRIEL SOARES DE SOUZA: *Tratado descriptivo do Brazil em* 1587. Published in *Rev. Inst. Hist. e Geogr. do Brazil*, T. XIV, 1851. The passage will be found on page 241 of this printed version.

G. SOARES DE SOUZA, who went to Bahia in 1570 and there became a planter (*roçeiro*), presented his manuscript to the Portuguese Government. Copies of it were in existence in Paris, Madrid, Portugal (Evora, Oporto, and Lisbon), in Brazil, and at Neuwied. The work was not printed till 1825, when it was published by the Royal Academy of Sciences of Lisbon. It appeared in 1851 in Brazil, through the efforts of F. A. DE VARNHAGEN.

† LUTZ, ARAUJO, and FONSECA (filho): Viagem scientifica no Rio Paraná, etc. *Mem. Inst. Oswaldo Cruz.* T.X. fasc. 2, 1918, p. 127.

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under the name of *boubas* in the Kiswahili language (VAN NITSEN), and at Zumbo (Mozambique) it is called by the same name (CAPELLO and IVENS). It seems probable that the name *boubas*, and the disease to which it is applied, are both of African origin.

Although WILSON (1868) recorded that it was believed in the West Indies that frambœsia is carried by flies, it is only in recent times that the conditions necessary for its transmission have begun to be understood. HIRSCH (1898) gives a description of two cases in which the disease appeared to him to have been conveyed by flies. Both patients were living with children suffering from frambœsia, and both presented "raw surfaces which could be inoculated by the poison." CADET (1897) considered that infection can only occur through pre-existent cutaneous lesions.*

But it was not until 1907 that CASTELLANI† demonstrated experimentally that flies can transmit *Treponema pertenue*. He fed a certain number of flies (*Musca domestica* and allied species) on scrapings from the slightly ulcerated papules of patients suffering from frambœsia; and on examining twenty-five of these flies subsequently, he found *T. pertenue* present in preparations made from four of them. He then placed flies, contaminated in a similar manner, on the deeply scarified eyebrows of twelve monkeys, and succeeded in thus infecting three of these animals.

It would thus appear that our GABRIEL DE SOUZA clearly understood the part played by non-biting diptera in the transmission of frambœsia, while he accurately defined the condition necessary for infection—the previous existence of an excoriation of the skin, upon which the fly deposits the virus. One would like to know to what little non-bloodsucking dipteran, of diurnal habits, the name *nhitinga* was applied; but I have not been able to elucidate this question.

It will be seen that GABRIEL DE SOUZA held views far in advance of his time. He might well have written, indeed, a recent chapter on the mechanism of infection, and even on the prophylaxis, of frambœsia—thus

*All these authors (WILSON, HIRSCH, and CADET) are cited by Professor G. H. F. NUTTALL in "The Part played by *Musca domestica* and allied (non-biting) Flies in the Spread of Infective Diseases." *Reports of the Local Government Board on Public Health and Medical Subjects.* New series, No. 16, 1909.

†A. CASTELLANI: "Experimental investigations on Frambœsia tropica (Yaws)." *Journ. Hyg.*, VII, 558, 1907.

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anticipating modern views by three centuries. His name is one, therefore, which should henceforth figure in the history of tropical medicine.

Collares. *February*, 1921.

ADDENDUM.

Professor CLIFFORD DOBELL, F.R.S., to whom I wrote on discovering the passage cited above, has had the kindness to inform me that J. F. X. SIGAUD, in his work *Du Climat et des Maladies du Brésil* (Paris, 1844), refers to a manuscript which is in the Royal Library at Rio de Janeiro, and which treats of framboesia (yaws or pian) and is dated 1587. Professor DOBELL was struck by the correspondence of the date and subject with those of DE SOUZA'S manuscript; and it is, indeed, a very curious coincidence that there should be in existence two such manuscripts dealing with framboesia and both bearing the same date (1587). The manuscript of which SIGAUD knew, however, does not mention contagion by diptera; but it is, nevertheless, very interesting. It records how general the "mal de bobas" was among the Tupinambas, and notes the prevalence of the disease among children, and the native methods of treatment; and, furthermore, it leads one to the conclusion that framboesia was already distinguished from syphilis towards the middle of the sixteenth century. See "*Descripção geographica da America portugueza, 1587.*" Chap. LXVII, p. 186.

A printed copy of this very rare work exists in the Public Library at Lisbon. According to some manuscript notes inserted in it, it appears that it was originally printed prior to 1811, the year of its actual publication; but its issue was suspended owing to a dispute arising between the editor—the botanist, F. J. M. DA C. VELLOSO (1742-1811)—and the Minister in charge of the Royal Stationery Office (*Regia Officina Typographica*). The original manuscript was subsequently discovered, almost complete, in the Royal Library at Rio de Janeiro.

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EXTRACTS FROM THE TROPICAL DISEASES BULLETIN.

The Council has decided to publish, with the agreement of the Tropical Diseases Bureau, extracts from the *Tropical Diseases Bulletin*, not exceeding four pages in each number of the TRANSACTIONS. Colonel S. LYLE CUMMINS, C.B., C.M.G., has kindly offered to select these for publication, and the first series appears below.

MEBANE (TOM S.). **Diagnosis and Treatment of Amœbic Abscess of the Liver.**—*Jl. Amer. Med. Assoc.* 1920. Dec. 4. Vol. 75. No. 23. pp. 1566-1567.

These observations are based upon a careful study of fifty cases of liver abscess treated at the Santo Tomas Hospital, Panama, during a period of three years prior to August, 1917.

Rupture into other viscera occurred in the order of frequency into (1) the abdominal wall; (2) the subdiaphragmatic space; (3) the pleural cavity; (4) the lung, and (5) the intestinal tract. Liver abscess may give rise to several well-marked clinical pictures depending upon its location and site. The most characteristic localizing symptom is the marked tenderness elicited by tapping over the liver. Jaundice—particularly anything more than a slight scleral tinting—should be regarded as being against a diagnosis of liver abscess. Nearly all the cases showed a definite leucocytosis averaging 18,000, but varying from 12,000 to 30,000. There are cases which simulate chest conditions—usually the result of a large liver abscess located in the dome of the liver; the diagnosis may be further obscured by a pleural effusion consequent to irritation of the pleura. Usually the real condition can be detected by noting that the dulness does not follow the sinuous course of most pleural effusions—it is just in these cases that assistance can be procured from X rays.

Another type of case may simulate disease of the gall bladder, the symptoms of which are produced by an abscess on the under surface of the liver, together with secondary peritoneal adhesions.

The high mortality, more than a third, in the present series of cases could be ascribed to the poor condition of the patients on admission. As a means of arriving at earlier diagnosis—so very important—the more extended use of hepatic puncture is recommended. As a matter of precaution all punctures were made in the operating room with the patient prepared for operation. When pus was found immediate operation was performed. When possible, the transthoracic method of approach is to be preferred, the direction of the needle of the successful exploratory puncture being taken as a guide. When approach through the chest is impossible, a rectus, or Kocher gall-bladder, incision may be employed. P. H. M.-B.

STOKES (ADRIAN) & BIGGER (J. W.). **A Short Account of Dysentery in Dublin in the Autumn of 1919.**—*Dublin Jl. of Med. Sci.* 1920. Mar. Fourth Series. No. 1. pp. 3-8.

In August, 1919, an epidemic of bacillary dysentery, the first recorded in modern times, broke out in Dublin. In all, 186 cases with 19 deaths were notified. The

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pathological anatomy in the autopsies was characteristic of bacillary dysentery. Out of 139 patients, whose fæces were examined bacteriologically, the dysentery bacillus was isolated in 76, or in 59·6 per cent. The majority were Shiga infections; Flexner's bacillus was encountered once only. Serological reactions were found to be most regular; the highest titre with the homologous organisms being 1 : 80. No difficulty was experienced in isolating or identifying the organisms, and a rough method of preliminary agglutination of the colonies with specific antiserum was employed.

P. H. M.-B.

KIRSCHNER (L.) & SEGALL. **Zur Bakteriologie der Ruhrerkrankungen des Jahres 1920 in Wien.**—*Wien. Klin. Woch.* 1920. Dec. 23. Vol. 33. No. 52. pp. 1125-1126.

The experience of the authors was gained in the 1920 epidemic of bacillary dysentery in Vienna. Impressed with their failure to isolate dysentery bacilli from a large number of fæcal specimens when transmitted to the laboratory, as well as by reports of FRIEDEMANN, who claims to have obtained positive results in 100 per cent. of his cases by means of rectoscopic culture [see this *Bulletin*, Vol. 11, p. 259], they decided to give the latter method a trial. From stools transmitted to the laboratory positive results were recorded in only 6 per cent.; from freshly passed fæces cultured in hospital the figure was 39 per cent.; while from mucus obtained by the proctoscope successful isolations were recorded in 65 per cent. Examples of the more familiar types of dysentery bacilli—Shiga, Flexner Y, Strong's bacillus—and also in certain cases SCHMITZ'S recently described type were recognised. They also suggest that *Bacillus fæcalis alcaligenes* plays a rôle in producing dysenteric symptoms.

P. H. M.-B.

RELE (V. G.) **Incidence of Sprue in Bombay, its Pathology and Treatment. A Paper read before the Bombay Medical Union on Jan. 16, 1920.**—37 pp. Bombay. 1920. Printed at the Bombay Vaibhav Press. [Price Two Rupees.]

The author has contrived to say a good deal about sprue in a reasonably short space. It is impossible to obtain exact statistics about the mortality in sprue in a city such as Bombay, where the monthly mortality from all causes is about 2,500. The disease, however, has assumed a position of considerable importance in this city, for which the climate and conditions of life are held to be responsible. Sprue is usually a disease of mature years, being common between the ages of 25 to 50, and is rarely seen in children. It is commoner in women than in men; in fact, women in Bombay, according to the author's experience, have a higher ratio of mortality from intestinal diseases of all kinds than have men.

The endemicity of sprue in Bombay is not held to be due directly to climatic conditions, because in other towns of the Presidency similarly located the disease is by no means so common, nor can it be held to be due to the food, as other communities, who live on exactly the same dietary, escape. Though sprue cannot be said to originate in unsanitary conditions, it finds a very congenial soil in places where drainage is neglected. The author is inclined to believe that sprue is nothing but a biological manifestation and final stage in the progress of dyspeptic trouble, and is the result of prolonged over-secretion by the stomach and duodenal glands.

The occurrence of one or more cases in the same family leads one to suppose that infection from one sprue case to another is possible. The symptoms of sprue group themselves into three stages:—

First stage, oral and dyspeptic troubles.

Second stage, intestinal symptoms.

Third stage, toxæmia, inanition, and anæmia.

Sprue may be grafted upon acute bacillary dysentery, or any other intestinal complaint, and in that case may prove fatal in a very short time.

The section on the pathology is purely speculative. It is thought that all the

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changes observed in sprue hinge upon the deficient production of secretin. Deficiency of this substance leads to a lowered activity of the pancreas, with consequent deficient digestion and mal-assimilation of food.

With reference to treatment, he is by no means confident that chromosantonin, as advocated by BEGG, justifies the claims made for it. In his practice he considers that in one case the drug was responsible for a smart intestinal hæmorrhage, and in a second for acute jaundice with high fever. Emetine injections are stigmatized "as a waste of a good remedy for what it is not meant." A milk dietary in Bombay being unsatisfactory, recourse should be had to treatment with buttermilk. One teaspoonful of curds is added to 26 oz. of milk previously boiled and cooled. After curdling it is churned and water added. The fat must be removed from the curdled milk, and churning should not be considered complete until this has been effected. The resulting butter-milk is given in much the same manner as the milk treatment is usually arranged. The patient is told to take 38 oz. of milk a day for curdling, and the next morning is instructed to take his first cup at 7 a.m. and the remaining quantity he is to finish before 3 p.m. After that time he is to take the milk prepared from that set aside early in the morning of the same day, and so on. During the night-time nothing but water is drunk. The idea is that, if kept more than six hours on a hot day, the butter-milk may turn sour. The amount is gradually increased, so that he is taking 70 oz. on the fifth day; finally, the maximum quantity in the 24 days should be 140 oz. (or seven pints). A pinch of table salt to a cupful of this milk greatly improves the taste. It is not possible to keep the patient on a regimen of this sort for more than two or three weeks, after which the addition of fruit is advisable. There are better fruits in Bombay than strawberries, which are very expensive. The following are recommended: pomegranates, sweet lemons (Santra), oranges, bananas, papaya, mangoes, apples, pineapples, peaches, pumelo, and musk melon. Pomegranates are specially recommended, and next to these bananas—whether dubbed locally "plantain" or "banana" does not signify as long as they are ripe. Some patients do well on mango juice. Although most cases do well on the butter-milk diet, there is a group which is in no way benefited. They go on losing in weight, and the doctor is blamed for starving them; these are hopeless cases, beyond the range of any form of treatment.

The author does not consider a change of climate necessary as long as the patient carries out the treatment effectively at his place of residence. The seaside is better than the hill station for sprue convalescents in India. A good rule to observe is to send patients away from the endemic area to places where the mortality from diarrhœa and dysentery is lowest. P. H. M.-B.

CONRAN (PHILIP). **Meat Diet in Sprue.**—*Brit. Med. Jl.* 1920. Aug. 7. pp. 206—207. With one chart.

A pure meat diet, almost the complete antithesis to milk regime, is occasionally of value in the most advanced and chronic stages of sprue. Two cases illustrating the advantages of this method of treatment are cited in the paper under review. The first case contracted sprue in India. At the beginning of 1919 she weighed only 5 stone 8¾ lbs., and presented signs and symptoms of an advanced stage of the disease; there was considerable anæmia. Test meals showed that there was general delay of digestion and assimilation throughout the intestinal tract. Meat diet consisted of 1 lb. 9 oz. of minced beef daily, pressed into cake and lightly cooked with 2 oz. of suet, and taken as five meals of 5 oz. each. After a few days the quantity was increased to 1 lb. 14 oz., given in five meals of 6 oz. each; hot water being sipped between each feed.

Subsequently this diet was considerably amplified by the addition of strawberries, egg-flip, bananas, rusks, etc., and in the nineteenth week the number of meals was reduced to four daily.

The dietetic treatment was supplemented by intestinal lavage, which proved of

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value in relieving the irritation of the lower bowel; in the later stages massage and the faradic current helped to restore the muscular tone of the abdominal wall.

During the first few weeks the weight dropped, and tetany of the hands and arms supervened. Medicinal treatment was for the most part symptomatic, chromosantonin grs. 5, twice daily, as recommended by BEGG, having little, if any, apparent effect. Improvement on meat diet was striking; the blood circulation first showed improvement; then the stools. In the last four weeks of the patient's stay at Duff House the average total day weight of fæces was 89 grammes, and over 4,000 calories of food were being taken.

On leaving the institution the patient had put on 2 stone 12¾ lb., and the bowels were opened naturally and regularly once a day on an approximately normal diet.

The second case, also a woman, contracted her illness originally in the West Indies, and gave a history of having suffered from two attacks of tetany in 1917. On admission her weight was 5 stone 12½ lb., together with considerable anæmia and the more familiar symptoms. The absorption of fat and protein was found to be deficient. With the introduction of an exclusive meat diet definite improvement began; the attacks of tetany ceased, and the condition of the mouth became normal. She was discharged from Duff House after eleven months' residence, and after having gained 1 stone 8¼ lb. in weight since admission. P. H. M.-B.

HEATON (TREVOR). **The Etiology of Sprue.**—*Indian Jl. Med. Res.* 1920. Apr. Vol. 7. No. 4. pp. 810–839.

This is a well-considered compilation originally submitted as a thesis to Oxford University in 1919.

It contains few original observations on the disease, but the information is well arranged.

The following are the conclusions drawn:—

Sprue is a definite entity, and not a mere association of symptoms. Its predisposing and complicating factors lead to, or depend upon, a single pathological condition.

There is no positive evidence to suggest that this is a specific bacterial or protozoal infection. These organisms may be present, and their destruction may be necessary for the cure of the disease; but sprue symptoms may exist in their absence.

The symptoms of sprue are due mainly to two factors, namely, mycotic fermentation and digestive incompetence. The two most probable theories of the disease are accordingly that it is a mycosis, or that it is a physiological exhaustion-state due to climate. Whichever of these be the primary, they operate in a vicious circle. Defective digestion favours the multiplication of organisms, while an infective catarrh accentuates digestive weakness.

The peculiarities of the incidence and geographical distribution of sprue point to a physiological rather than an infective cause.

The immediate relief from all infective systems in response to non-specific treatment is contrasted with the very slow and uncertain recovery of the digestive function. This is also taken to indicate that the digestive failure is the essential disability, and the infection secondary.

The prominence of infective processes in the mouth in most sprue cases, and their tendency to relapse, cannot be explained by any theories of physiological failure of digestion. It is, however, less easily explicable by any infective hypothesis, for it is in origin physiological, depending essentially upon a condition of lowered resistance; the actual infecting organism being accidental.

The physiological disability of sprue is brought about by the effects of climate. In cœliac disease of children a similar disability is present, but it is congenital.

Hill diarrhœa is essentially a form of sprue, acute in onset and uncomplicated in course; the disease affords an example of sprue symptoms arising in the absence of infective causes. The physiological fault underlying these three diseases may lie in the actual digestive glands themselves, the liver and pancreas. Possibly, however, it may be that the supply of the chemical ferment secretin is deficient, thereby depriving these glands of their normal stimulus. P. H. M.-B.

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Proceedings of a meeting of the Society held on Friday, June 17th, 1921, at 8.45 p.m., at 11, Chandos Street, Cavendish Square, W. 1, Professor W. J. R. SIMPSON, C.M.G., M.D., F.R.C.P. *(President)*, in the Chair.

ANNUAL GENERAL MEETING.

The PRESIDENT: Gentlemen, you have before you the Report of the Council for the year ending June, 1921, and those who have read it will, I am sure, agree with me that it is a very satisfactory one. The progress of the past year is, at least, equal to that of the preceding years. There have been difficulties, which were common to all societies in the time of stress, but they have been successfully overcome. This is due to the unremitting attention paid to the Society's affairs by Dr. LOW, as mentioned in the Report, and our present Hon. Secretaries, as well as to our Treasurer, Dr. BALFOUR, who is at present in Mauritius. The Society owes a debt of gratitude to these gentlemen. The status of our Society has been improved, and we have also maintained our individuality and independence. There was at one time a movement, proceeding from the Royal Society of Medicine, for us to join that Society as one of its Sections. But we felt that our work in the tropics was of such importance that it was best for us to remain separate, and to concentrate our attention on these matters. even in preference to joining such a distin-

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guished body as the Royal Society of Medicine. Now that that danger has passed, the Royal Society of Tropical Medicine and Hygiene will be willing to co-operate with the Royal Society of Medicine in any movements or discussions which have for their object the improvement of the health of the tropics.

The next point is this: that we are now the Royal Society of Tropical Medicine, and it is very unusual for so young a Society to become a Royal Society. I attribute this to two causes: the first is, that the Fellows, who are living in all parts of the tropical world, have presented to this Society very valuable papers on the causes, treatment and prevention of tropical diseases. And I attribute it also to the desire on the part of His Majesty the King, who has visited the tropics and seen the conditions there, to encourage the prosecution of the study of Tropical Medicine and Hygiene in our Possessions.

Another feature has been that, just as we have become a Royal Society, there has been presented to us by Mrs. CHALMERS, in memory of her husband, who was a Fellow of this Society, a gold medal. His death we deplored; he was just coming home, and we hoped to have the benefit of his very varied experience in West Africa, in Ceylon and in the Sudan at our counsels. I know he took a great interest in the Society.

As is well stated by the Secretaries, we should now look forward to endeavouring to get a home worthy of the Society. Unfortunately, trade depression and high rentals and high prices of materials, coupled with heavy taxation, make it obvious at the present time that we shall have a difficulty in finding those premises which we want. Still, I think there are hopes, and when this depression has passed away I am sure we shall ultimately get a good home. In the meantime we are housed, thanks to the Medical Society of London, in this comfortable and historic building; and we have, in that respect, owing to its central situation, a very great advantage.

With these remarks I propose that the Report, as printed, be adopted by this Society. Perhaps one of the Fellows will second that motion.

The Report was seconded and carried *nem. con.*

The PRESIDENT: The next item is the presentation of the Treasurer's Statement.

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Dr. A. G. BAGSHAWE: In the absence of Dr. ANDREW BALFOUR, I will just say a word or two about that Statement. It shows that, as far as finances go, we have had a successful year. We carry forward a balance of £294 odd. The accounts have been audited, for the first time, by a Chartered Accountant, and some extracts from his report are given. I move the adoption of the Treasurer's Report. (Seconded and carried.)

The PRESIDENT: The next business is to announce the result of the ballot for the President, Vice-Presidents and Council:—

President:

Sir JAMES CANTLIE, K.B.E., F.R.C.S.

Vice-Presidents:

ANDREW BALFOUR, C.B., C.M.G., M.D.

Surg.-Rear-Admiral Sir P. W. BASSETT-SMITH, K.C.B., C.M.G., F.R.C.S., R.N. (retd.).

Professor J. W. W. STEPHENS, M.D., F.R.S.

Council:

Col. A. ALCOCK, C.I.E., F.R.S., I.M.S. (retd.).

Major E. E. AUSTEN, D.S.O.

A. G. BAGSHAWE, C.M.G., M.B.

Surg.-Cdr. R. ST. G. S. BOND, R.N., M.B., M.R.C.P.

Lieut.-Col. WM. BYAM, O.B.E.

CLIFFORD DOBELL, M.A., F.R.S.

H. M. HANSCHELL, D.S.C., M.R.C.S.

Lieut.-Col. S. P. JAMES, I.M.S. (retd.).

Lieut.-Col. J. C. KENNEDY, R.A.M.C.

Col. W. G. KING, C.I.E., I.M.S. (retd.).

J. C. G. LEDINGHAM, C.M.G., M.D., F.R.S.

G. C. LOW, M.D., M.R.C.P.

P. H. MANSON-BAHR, D.S.O., M.D., M.R.C.P.

H. B. NEWHAM, C.M.G., M.D., M.R.C.P.

G. BASIL PRICE, C.M.G., M.D., F.R.C.P.

A. LESLIE SHEATHER, B.Sc., M.R.C.V.S.

H. S. STANNUS, M.D., M.R.C.P.

J. D. THOMSON, M.B.

C. MORLEY WENYON, C.M.G., C.B.E., M.B.

WARRINGTON YORKE, M.D.

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Treasurer:
G. C. LOW, M.D., M.R.C.P.

Secretaries:
C. MORLEY WENYON, C.M.G., C.B.E., M.B.
G. BASIL PRICE, C.M.G., M.D., F.R.C.P.

I now have the pleasure of introducing the new President, Sir JAMES CANTLIE, to preside over you for the coming two years. Sir JAMES CANTLIE really does not require any introduction, but I may say he was one of the founders of this Society, and so it is very appropriate that he should become its President. He has been the founder of many things. I know personally that he was one of the founders of the London School of Tropical Medicine; I know, also personally, that he was the founder of the *Journal of Tropical Medicine*; in fact, he can never rest, he is always founding something. There is one thing which I very much hope he will found during his presidency, and that is that he will be able, by his energy and his persuasive ways, to secure a home worthy of this Society. I have now much pleasure in asking Sir JAMES to take the Chair.

Sir JAMES CANTLIE then occupied the Chair.

The PRESIDENT (Sir JAMES CANTLIE): Professor SIMPSON and Gentlemen, it is a very great honour you have paid me. After the very kind things Professor SIMPSON has said, and the kind way in which you voted to place me in this position—a very important one—I assure you I will do my best, while I am here and while I am fit, to continue to attend the meetings with punctuality and regularity; that is to say, I will do my best to regenerate in my old age.

This Society, as Professor SIMPSON has said, was founded in my old house, 140, Harley Street, by a number of you who are here present. Of course, we could not get on without Sir PATRICK MANSON, and he was always in my mind; and afterwards, Dr. CARNEGIE BROWN, who was a retired man and had a large amount of money behind him. He took the secretaryship, and the Secretary at that time had to do much labour, and he must have had a good deal of free time, otherwise he could not have done the work. And I chose the men who, I thought,

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should fill the various posts. Dr. CARNEGIE BROWN we lost—a serious loss both for this Society and for other reasons.

I hope we shall be able to get enough money for a place for this Society, and I think we shall. I have got into the way of raising money. Perhaps you may find I have not been so successful as one might have been, but that this Society deserves support and should have a place of its own there is no doubt. I think that, with our combined efforts, we shall have a place of our own, and if we do not I shall die unhappy, because it will be the first thing I have tried and not succeeded in.

I thank you very sincerely for the very great honour you have done in placing me in this position to-night.

The PRESIDENT: Our next business is to elect the Auditors.

Dr. BAGSHAWE: The Auditors for last year were Dr. BEDDOES and Dr. MANSON-BAHR. Dr. MANSON-BAHR has just become a member of Council, so we cannot ask him to serve in this post of auditor. I propose that we ask Dr. BEDDOES to serve for another term, and Colonel CLAYTON-LANE be nominated as his colleague, both for two years. (Seconded and carried.)

The PRESIDENT: We now come to the resolution for the alterations of the Laws.

Dr. C. M. WENYON: When the question of election of new Councillors came up some time ago, it occurred to me that it might be better to make provision in the Laws for bringing about a change in the Council at the biennial elections. I suggested it would be well to have a Rule whereby not more than fifteen of the twenty old Councillors should be re-elected so that we should secure a change of blood on the Council, which would probably help us to have new ideas for carrying on the work of the Society. Accordingly I propose that Rule 4, which reads as follows: "Councillors shall be elected every two years in the manner hereinafter provided," shall be altered by adding the words, "but not more than fifteen of the twenty shall be re-elected."

The resolution was seconded by Major E. E. AUSTEN, and, after some discussion, carried unanimously.

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The PRESIDENT: I have the greatest pleasure in proposing a hearty vote of thanks to Professor SIMPSON. He has done, in many countries, excellent pioneer work as a public health man, among all the talent of Tropical men in different branches of thought and work. You all know the work he has done—it is not necessary for me to tell you—in South Africa, in West Africa, India, and various places in the world. And there is no man in the world who can write a report like SIMPSON on sanitary matters. He has now completed his excellent work by his presidency of this Society during the past two years.

Professor SIMPSON, I convey to you, Sir, our very sincerest thanks. You have raised us from the Commons to the House of Lords, and even beyond that, for we have been brought to the very Throne itself; we are now Royal.

Sir LEONARD ROGERS: I had the pleasure of knowing Professor SIMPSON in Calcutta twenty years ago, when I was there as a young man, and he showed me great kindness and took an interest in one of my papers, mentioning me to the Director-General, and that gave me an opportunity of investigating kala-azar. We lost him soon after I got to Calcutta, and I did not see him much again until I came home. I have great pleasure, gentlemen, in endorsing all that has been said by the Chairman.

Professor SIMPSON: Gentlemen, I thank you very much for the way in which you have received this proposal.

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THE MECHANISM AND TREATMENT OF SNAKE-BITE IN INDIA.

BY MAJOR R. KNOWLES, I.M.S.

When the Secretaries did me the honour of asking me to read a paper before the Society, it seemed to me that possibly it might be of interest to members if I attempted to give you some résumé of the work carried on from 1912 to 1914 by Major ACTON, I.M.S., and myself, at the Pasteur Institute, Kasauli, on the problem of snake-bite in India. In doing so, however, it will be necessary to make such report as concise as possible, since the ground to be covered is enormous.

The term "snake-bite," in reality, includes more cases of bite from non-poisonous than from poisonous snakes. The common Indian rat-snake, or "dhaman," is frequently mistaken for a cobra, and bites freely and viciously if disturbed. One of our tame pythons at Kasauli bit three of us in a month. The common and perfectly harmless *Lycodon auliculus*, or wolf-snake, is constantly mistaken for a krait. Of the many species of vipers in India, it is probable that not more than two, or possibly three, are fatal to man, although such snakes can readily kill birds and small animals. Even in untreated cases of true cobra-bite, for reasons which will follow, I do not think that the mortality in man can be placed at much over 40 per cent. Hence, although the annual mortality in India from snake-bite is placed at 25,000 per annum by the Sanitary Commissioner, it is probable that the total number of cases of snake-bite of all descriptions run into many more thousands, whilst the actual percentage mortality from such cases is not a large figure. It is upon this foundation of random sampling, and upon advertisement, that the so-called success of the thousand-and-one remedies for snake-bite in India really depends. A correspondent in Kashmir once sent us a root with

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which he claimed to have treated without a failure all cases of snake-bite seen by him in Kashmir in twenty-five years. The fact is, however, that the few poisonous snakes of Kashmir belong chiefly to the small and non-lethal vipers, and had this correspondent used ginger-beer or followed the more traditional practice of giving his patients so much alcohol that they saw again the snakes which bit them, the results would probably have been the same. The problem of how to neutralise successfully a lethal dose of colubrine or viperine venom after its injection into the human body, is one which not one of the many hundreds of remedies now in vogue in India really touches.

The lethal snakes of India may be divided into three groups. The first is the group of sea-snakes or *Hydrophineæ*. They occur in the salt-water marshes around the Indian coasts, and I have seen them in large numbers near the mouth of the Shatt-el-Arab in the Persian Gulf. They are large snakes with fine heads, thin necks, thick bodies, and tails flattened like the blade of an oar. The colouring is very brilliant, and usually in the form of broad transverse bars. Very little is known about their venom, although Sir L. ROGERS found it to be intensely toxic and with an intensely paralytic action on the central nervous system. We were not fortunate enough to secure sufficient sea-snake venom for experimental work; and, although some, at least, of the deaths "from drowning" among Indian fishermen on the Indian coasts are due to these snakes, yet they cannot be said to add materially to the death-rate from snake-bite in India.

The second group is the great group of the *Indian Poisonous Colubrines*: the cobra, the king cobra or hamadryad, the kraits, and the coral snakes. It is this great group which renders the group of Indian snakes so deadly. Non-poisonous colubrines abound everywhere in almost all continents, but it is in Asia and in Africa that these poisonous colubrines occur, and make the problem of the treatment of snake-bite so difficult. In Australia and South America, where the chief poisonous snakes are vipers, the problem of snake-bite is far less difficult than in India.

The third group are the *Vipers*. India is full of vipers; but only two—the Russell viper, or daboia, and the *Echis carinatus*, or saw-scaled viper—are really lethal to man. In three years, at Shillong, I have seen six cases of bite from the very common *Lachesis monticola*, or large spotted viper, and two from the common green pit viper, *Lachesis*

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gramineus, all without any symptoms of venom intoxication following. The small vipers very frequently bite man: they are very rarely, if ever, fatal. All vipers possess venom glands and specialised fangs, but in only two of the Indian species, probably, is there present sufficient venom of sufficient potency to cause death.

Our work is divisible into four main sections, with which I will endeavour to deal in turn as rapidly as possible. The first question which we set ourselves to answer was this: Under natural conditions, and at a good bite, what doses of venom do the really lethal snakes of India inject into man? There had been some previous work on this problem, but we felt that it was vitiated by the very small numbers of observations and by the unnatural conditions of experiment observed. If there is one thing more than another needed in modern medicine it is the collection of sufficiently large numbers of observations before deductions can safely be made. It is not, in reality, the exception that proves the rule, but the rarity of the exception. A single observation may be of importance, but is open to doubt. Ten similar observations suggest uniformity; a hundred amount to a probability; 500 to 1,000 render the probability almost a certainty. As our work was bound to be almost entirely experimental, we decided to try and pile up dozens or hundreds of experimental observations on each point before coming to conclusions. In place of a few random experiments on the usual type of laboratory animals, we utilised hundreds on rats. The rat is a most suitable laboratory animal for large observational purposes. It is easily kept, easily fed, healthy as a rule, and can be easily handled with gloves and a pair of large forceps. Twice a week we received large consignments of rats, caught in connection with the rat-catching operations at Delhi; whilst the hundreds of poisonous snakes utilised in the three years were caught for us by three gangs of professional snake-catchers in the jungles of Behar, and railed in weekly batches to us by Mr. Reid of Champaran. Thus we had available almost unlimited material for observational purposes: thousands of healthy rats and some hundreds of fresh, active snakes.

We began with desiccated cobra venom. Now desiccated cobra venom is almost as stable and as lethal a substance as a lethal alkaloid. Colubrine venoms kill, as I need hardly remind you, by causing paralysis of the respiratory centres. In the case of cobra bite, death is heralded by asphyxial convulsions; in the case of common krait venom the

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complete paralysis of the body is more marked, and the asphyxial convulsions less in evidence. But all colubrine venoms kill really by causing respiratory failure, and in experimental work we may take the last respiration as the death point.

To each of 465 adult healthy rats we gave hypodermically 5 mgms. of desiccated cobra venom in solution in normal saline, and in each case timed the interval to death in minutes and seconds. Now 5 mgms. of cobra venom may kill a rat in as short a time as 10 minutes, or it may take 65 minutes; but the mean of these 465 observations works out at exactly 30 minutes with a very small standard deviation. If, per contra, a rat which has received an unkown dose of cobra venom dies in 30 minutes it does *not* follow that the dose of venom given was 5 mgms.; but if a large series of rats after receiving unknown doses of cobra venom die on an average in 30 minutes, it follows that the average dose given was about 5 mgms. of cobra venom.

With the 5 mgm. point on this curve determined, we then performed similar series of experiments at 1, 2, 3, 10, 20, 30, 40, 50, 60, 70, 80, and so on to 600 mgms. of venom, with a total of 864 observations finally placed on the curve. In this way we obtained the experimental dose-death-time curve shown on the Chart.

This curve at once presents two interesting theoretical features. In the first place if we give a rat a dose which is not sufficient to kill, in other words a sub-M.L.D., then the rat will survive, and its death time becomes infinity; in other words, at its commencement the curve runs parallel to the vertical axis. To determine this point accurately we determined the M.L.D. of cobra venom for the rat, and from 54 observations found it to be one-tenth of a mgm., and at this point the curve becomes parallel to the vertical axis.

Secondly, whether one gives a rat hypodermically ten lethal doses or 10,000 lethal doses of venom, it cannot die until one M.L.D. has been absorbed into the circulation; and, at what we may term the "absorption period of cobra venom for the rat," the curve becomes parallel with its horizontal axis. The mean of twelve such observations, conducted with massive injections of venom, worked out at just under three minutes, and at this point the curve becomes parallel with its horizontal axis.

Now a curve of such character, lying between rectangular axes, and parallel at each end to an axis, suggests a rectangular hyperbola; and we

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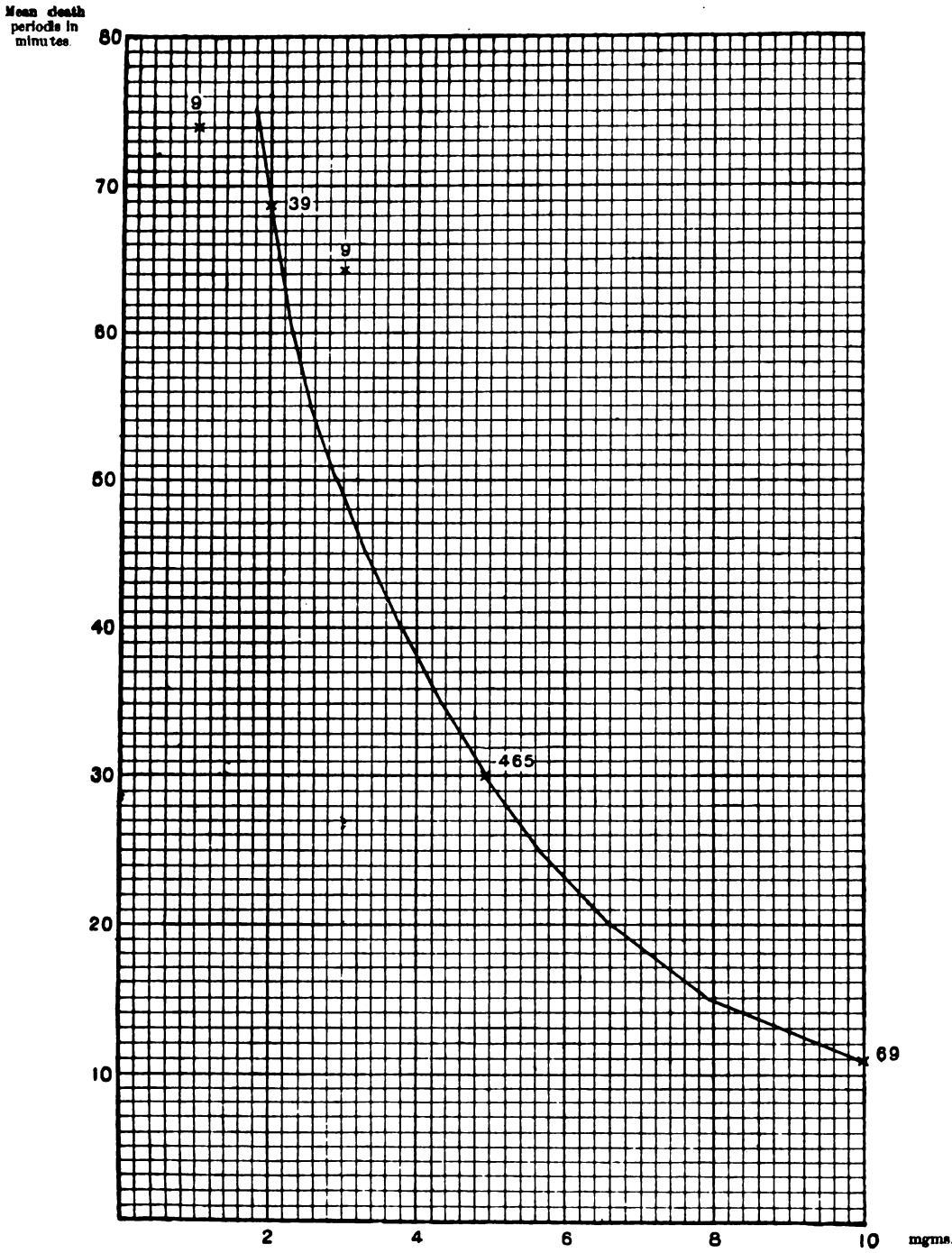

Showing mean death periods of 582 rats which received from 1 to 10 milligrammes of Cobra venom hypodermically. The numbers on the curve are number of observations at each point.

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found that our experimental curve, obtained from the means of 864 observations, coincided almost exactly with a regular hyperbola with a regular equation to represent it. In determining the dose given at a bite, we preferred the use of the equation as being more accurate for larger doses; but either experimental curve or equation give almost identical results.

We now proceeded to make cobras bite rats under conditions approaching those in nature as nearly as possible. The result of our first experiment was as amazing as it was unexpected. I know that it sounds quite incredible, but what happened was that the rat killed the cobra. A large fresh monocellate cobra and a large freshly captured rat were placed together in a large box, with ample room for movement. The cobra reared with expanded hood and struck downwards at the rat. As he did so the rat jumped up into the air, landed near the snake and bit it. The battle lasted for fifteen minutes, after which the combatants were separated and separately caged. Next morning the cobra was found dead from shock and showed fourteen wounds. The rat, which was untouched and in good health, was given his liberty. This anomalous result was probably due to battle taking place by daylight.

Accordingly, after this experience, we had to hold both the cobras and the rats, and in turn to present the hind limb of each rat to the biting snake. In this way we collected 58 first observations, in which one fresh cobra in each case bit one rat; and 115 second and subsequent observations, in which each snake bit from two to seven rats in succession. The mean of the first series of observations worked out at 172 mgms. of desiccated venom, and the actual doses given ranged from 3 to 360 mgms. of desiccated venom. In the second and subsequent strikes the doses given ranged from nil to 360 mgms. In the case of every cobra the amount of venom remaining in the glands after biting was expressed, collected, dried and weighed. Eighty-nine fresh cobras were used in these experiments. With 18 other cobras, which had been kept in captivity for some 16 days to 4 weeks, we obtained a very different figure: an average first bite of only 61 mgms.

Now there is one check on these results possible. The cobra gives, at a good bite, about two-thirds of the total venom content of the glands. Accordingly we milked 50 fresh cobras of every drop of venom in their glands: and two-thirds of the mean total yield of 317 mgms. gives a figure of 211 mgms.

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We may conclude from these two sets of experiments that, at a good bite under natural circumstances, a cobra can inject an average dose of from 172 to 211 mgms. of desiccated venom (the latter figure being probably the more trustworthy) whilst the actual figures obtained show the widest possible range of variation. One of our cobras gave a total venom yield of 881 mgms.—an enormous figure, which corresponds to a biting value of about 587 mgms. or about forty times the M.L.D. for man.

This extreme range of variation in the doses given is puzzling, until one comes to look into the mechanism of the bite, and this is best understood from actual dissection in the cobra and in the Russell's viper as representing respectively a poisonous colubrine and a viper.

Snake venom is, in reality, a modified saliva. The venom glands are modified parotid glands, and are situated in a corresponding position. From each venom gland there passes forward a venom duct, which opens at the orifice of the venom papilla at the base of the fang. In the colubrine the fangs are short and fine.

The cobra can only strike in a downward and forward direction and has to raise the head and expand the hood before a strike can be made; reasons which account for the fact that the cobra is the most easy of all venomous snakes to handle, since its every movement can be watched and anticipated. Further, during the daytime the cobra is often very disinclined to bite, and quite frequently will make simply a strike with closed mouth, with the intention of merely frightening its opponent.

When at rest the fangs in the cobra's mouth are directed backwards towards the pharynx. As the head is raised and the neck expanded, the mouth is very widely opened, the fangs rock into a position consequently in which they point forwards, and the mucous membrane covering the base of the fang is retracted (the vagina dentis), so that the fang is bared. As the fang rocks forward the base of the venom canal in the fang is brought into accurate apposition with the venom orifice in the venom papilla.

The snake now strikes downwards and forwards at its prey, and buries the fangs in the flesh.

Now the venom gland in the colubrine lies between two masses of muscle. One passes from the mandible and is inserted into the posterior and inner two-thirds of the gland surface. The other passes from the

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temporal region to be inserted into the posterior and outer two-thirds of the gland surface.

And the most essential feature of the bite with the poisonous colubrine snakes is that the *lower* jaw, as well as the upper, must be closed before the snake can inject any venom at all. As the fangs penetrate into the flesh, these two masses of muscle contract, and the venom gland which lies between them in a lymph space is compressed. Owing to the character of the insertions of the muscles the action which they exert on the gland is exactly like that of wringing out a wet towel, and acts only on the posterior two-thirds of the gland. Hence two-thirds of venom content of the gland is expressed out and along the venom duct. It passes through the venom orifice into the venom channel of the fang, and is deposited in the flesh; not exactly under the site of puncture of the fangs, but at the spot to which the curved ends of the fangs have reached in the subcutaneous tissues.

It is obvious that all this represents a complicated and a very highly organised mechanism. It is also obvious that such a complicated mechanism may easily go wrong. In fact, there are ever so many causes which may lead to a cobra failing to give a lethal dose of venom at a bite. During the daytime the cobra may strike with closed mouth and with no intent to bite at all. Even if biting, it may mistake the distance of the strike in daylight and fail to really bite. The sudden start on the part of the animal struck at may lead to the same result. The fangs may become entangled in fur or clothing and fail to lodge in flesh. They may merely graze the skin, in which case no venom is given. In nature cobras are sometimes met with whose fangs have been accidentally broken; and if such a snake bit, the venom would not be lodged but would merely dribble into its mouth. The approximation of venom orifice and venom canal may be inaccurate. Above and beyond all in importance in the case of the colubrine snakes, the snake when biting may fail to get in that firm closure of the lower jaw, without which compression of the venom gland cannot take place. In fact, you will see that there are numerous reasons why, even when a cobra has definitely struck a man, it may have failed to have given a lethal dose. One of our attendants was bitten three times by cobras in a year; in one instance the fangs merely grazed his thumb; a second time the man flung the snake off before it closed its lower jaw; on the third occasion the snake got in a

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true bite, but the cobra had been milked the previous afternoon, and had not got a lethal dose left in its glands. In not one of these instances did any symptoms of venom intoxication occur.

You will see, therefore, that the position with regard to the cobra is this. The dose given at a bite may vary from nil to 587 mgms. of desiccated venom, whilst an average dose at a good bite is about 211 mgms. or 14 to 15 times the M.L.D. for man. Further, the chances of escape, even in untreated cases of cobra bite, are so considerable that I do not think that the mortality amongst untreated cases can be put down at a much higher figure than 40 or 50 per cent. It is upon this fact that the so-called success of many so-called remedies depends.

The mechanism of the bite in vipers is different. There is nothing in nature more deceptive than a viper when about to strike. The snake lies on the ground coiled up, with its coils resembling one half of the mainspring of a watch, and the head resting in the centre of the coils. Nothing looks more sleepy. But the snake is very wide awake, and its eyes are following every movement of the prey; whilst the body alternately swells and contracts with a sharp hissing respiration.

Suddenly the snake strikes. There is no nonsense and there are no half measures with a viper. It is all out every time. The head darts upwards and forwards straight at its prey, and the action is so instantaneous and so sudden that it is often almost too rapid to follow. In all cases at least two-thirds of the snake's length leaves the ground, only the tail remaining fixed. In some instances I believe that possibly the snake may leave the ground entirely.

As the snake strikes its mouth is open to the widest possible extent, and an observer standing in front of its glass case can see right down into the pharynx. The fangs, which in vipers are enormous and very strongly recurved, are rocked forward by a mechanism similar to that for colubrines. So widely indeed is the mouth opened in vipers that the upper jaw would be dislocated at every strike were it not for the presence of a check ligament which prevents this accident. The snake strikes, the fangs lodge, and the closure of the lower jaw may or may not occur, but this point is of *no* importance in the case of vipers.

Now the venom gland in the viper lies between two flat strips of muscle which pass forward from the palate and pyterygoid region to be inserted into the maxilla.

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Once the fangs have penetrated into the flesh, almost every muscle in the snake's body comes into action. With the buried fangs acting as anchors, the head contracts down on to the neck, and the neck on to the body. If the prey be a bird or small animal, one can observe at this instant that it is often dragged right in towards the snake. With man the result of this muscular action is to bury the already lodged fangs to the hilt; in fact, a viper often has some difficulty in letting go after a good bite. At the same instant the venom gland is forcibly compressed between the two flat strips of muscle mentioned, and the entire contents of the gland are forced out with much the same action as one would employ in squeezing out the contents of a tube of tooth paste by pressure between the pages of a book. The venom is forced forwards and runs through the venom orifice and down the venom groove to the tip of the fang. If you will remember that the fangs of a full-grown Russell's viper are some three-quarters of an inch in length, and very strongly curved, you will see that the site at which the venom is actually lodged does not correspond to the site immediately underlying the fang punctures in the skin.

Not only is the mechanism of the bite entirely different in colubrines and vipers respectively, the method of action of the two classes of venom is also entirely different. I do not need to dwell on this point, because you are all familiar with it. But I may point out that colubrine venoms contain three toxic elements: a hæmolysin, which dissolves the red-blood corpuscles; a second element which actually acts as a cardiac stimulant; and a third lethal element—the neurotoxin—which paralyses the central nervous system, and causes death by paralysis of the respiratory centre. In man, colubrine venoms cause death from asphyxia in from thirty minutes to thirty hours after the bite.

Viperine venoms also contain three toxic elements: a hæmolysin; a second element which may be termed a coagulin, and which causes blood coagulation; and a third lethal element—a cytolysin or endolysin—which causes dissolution of the fine endothelial lining of capillaries and venules all over the body. Death after viper-bite occurs from cardiac failure in from two to seven days, but is due to a variety of causes. In the first place, the coagulant action of the venom may cause local coagulation in the vessels. In the case of small animals, if the fang happens to penetrate a vein, a clot may form, and may be swept into the

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circulation and kill immediately by pulmonary embolism, but I doubt whether such ever occurs in man. Secondly, as the result of extensive local coagulation, gangrene may set in and kill the patient. Thirdly, and more usually, sufficient venom is absorbed from the site of injection to bring into prominence the action of the cytolysin. The patient commences to bleed from all mucous membranes, and hæmorrhages occur both internally and externally. Then sepsis supervenes, and ultimately death from cardiac failure.

During the course of our work we spent nearly six months on a careful physical and chemical analysis of the two principal Indian venoms, and it is nearly impossible to summarise the results of this work in a few moments. One may however say this: venom is essentially a modified saliva, and, in addition to its lethal function, is also of great importance in digestion. People sometimes speak of the digestion of an ostrich, but the digestion of a snake is far more remarkable. In the case of the non-poisonous snakes, such as the rock python, one can easily observe how very thoroughly the snake salivates the entire body of its prey; and this is not merely to assist deglutition, for it is done as thoroughly with a small animal as with a large one. It is for purposes of pre-digestion, and snake venom has a similar function. Also venom is secreted in the same way as is saliva and by morphologically similar glands. One of our sets of experiments was to subject venom solutions to heat for five minutes at different temperatures, and then test the toxicity of such heated venoms. With cobra venom we found that the proteids coagulated at 65° C.; the hæmolysin was destroyed at 80° C.; but the venom still retained 80 per cent. of its original toxicity at this temperature. With viperine venoms the loss in toxicity on heating was more rapid, and such venoms are less thermostable. The cytolysin is destroyed at 85° C. Chemical analysis of venoms, and separation of the different proteid constituents of venoms, followed by testing each fraction for toxicity bears out the same conclusion: that the toxic principles in snake venoms are not of a simple character, but comprise a range of proteids and proteoses. With colubrine venoms the most toxic elements are the non-coagulable proteoses, and such venoms are relatively thermostable; with viperine venoms the toxic elements are more easily affected by heat. But no simple element containing all the full original toxicity was extracted in either class of venom.

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It is time, however, to return to our experimental dose-death-time curve. We followed up our experimental curve for cobra venom for the rat by the construction of a similar cobra venom curve for the monkey, *M. rhesus*, based, however, on only a few observations, and utilised only for crucial and control experiments. As the monkey gave a curve similar to that for the rat, but with different constants, viz., different M.L.D. and different absorption time, we concluded that for man also, such a theoretical dose-death-time curve for cobra venom could be obtained.

Assuming that there *is* such a theoretical curve for man we next set to work to study the whole of the literature, and from FAYRER'S records we extracted forty-eight fatal cases of cobra bite in which details were given, and in which no treatment of any value in delaying death can be said to have been applied. The absorption time for cobra venom for the rat we found to be just under 3 minutes; for the monkey it was found experimentally to be 7 minutes. In man it is therefore, probably, not less than 10 minutes. It cannot be much longer, because cases are recorded where symptoms clearly due to venom intoxication came on within 20 minutes of the bite. Now our extreme doses of cobra venom at a standard bite—or two-thirds of the total venom contents of the glands—were from 20 to 587 mgms. of venom, and the corresponding death periods in man are recorded by FAYRER as being from 30 minutes to 30 hours. Assuming that these figures must fit into a similar curve, which becomes parallel to its horizontal axis at about 10 minutes—that 20 mgms. kills in 30 hours, and 587 mgms. in 30 minutes—then we have all the factors necessary to calculate the equation for this curve, which was accordingly done. Further, at the point where this curve becomes parallel to its vertical axis we have the sub-M.L.D. of cobra venom for man, and this figure worked out at 13·6 mgms. In other words, the M.L.D. of cobra venom for man, calculated in this way from an entirely new standpoint, is about 14 or 15 mgms., a figure which agrees closely with that obtained by other workers.

I am afraid that I have dealt with this fundamental experiment of ours at some length. The remainder of the first section of our work followed the same lines. We constructed similar experimental curves for Russell's viper venom in the rat (based on 309 observations and shown on the screen); for the venom of the Echis viper (based on 117

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observations); determined the dose at a bite and the M.L.D.'s of different venoms for the rat and other experimental animals; also the venom yields of the different Indian poisonous snakes. In brief, as the result of eighteen months of experimental work, we were able to define the real meaning of the term, "bite from a poisonous Indian snake," in the following terms:—

1. The common cobra, both from the large amount of venom which it can give at a bite, and from its universal distribution is, *par excellence*, the poisonous snake of India. On the other hand many factors, including some peculiar to colubrines alone, render it certain that occasionally at least the cobra gives a sub-lethal dose.

2. The venom of the hamadryad (of which we were only lucky enough to secure two living specimens) is more toxic than that of the cobra. Its large size, its strength, its extreme agility, and its readiness to attack, probably render this colubrine the most lethal of all Indian snakes. It is, however, a relatively rare snake, except in certain areas.

3. The venom of the common krait is marked by its high degree of toxicity (indeed we found krait venom to be sixteen times as toxic to the monkey as is cobra venom). The dose given at a bite, however, is relatively small. This venom tends to cause death after a prolonged period, death being characterised by extreme paresis. The snake is thus an exceedingly lethal one, but, on the other hand its sluggishness and its disinclination to strike unless aggravated, lessen its importance in swelling the mortality.

4. The banded krait occupies an intermediate position between the cobra and the common krait, resembling, in doses given and natural disposition, the latter rather than the former.

5. The venom of the Russell's viper shows a marked tendency to produce local thrombosis and gangrene in such cases as escape death from hæmorrhage. The chances of escape after being bitten are considerable, but escape is often accompanied by severe gangrene and loss of the part bitten.

6. The venom of *Echis carinatus* shows less tendency to produce gangrene, whilst its hæmorrhagic action is very potent. Death usually occurs after a period of several days and is due to multiple hæmorrhages. On the other hand the doses of venom given by this snake are so small that the chances of escape in untreated cases are very considerable.

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7. The bite of the common green pit viper, and probably of all the Indian species of the genera Lachesis and Ancistrodon are rarely, if ever, fatal to man.

With so much by way of defining the term "snake-bite," we may turn to the second section of our work, which dealt with *the value of physical remedies* in snake-bite. Here it will be sufficient if I simply give the conclusions at which we arrived, as the experimental work covered the usual ground :—

1. With the exception of amputation, all measures of a physical character can only be classed as adjuvants to other forms of treatment.

2. The usefulness of ligature in poisoning from colubrine snakes is strictly limited. The table shown on the screen shows our main experiment on this point. The effect of ligature after cobra-bite is merely to prolong the interval to death. The injected venom is temporarily locked up within the ligatured limb. As soon as the ligature is removed, the imprisoned venom floods the circulation and kills the patient.

3. In viper-bites, however, and especially in the case of bites from Russell's viper, the value of an immediate and efficient ligature is very considerable. In some instances it may save life, although no other measure be employed. The table on the screen shows our main experiment on this point. At doses of from 5 to 20 mgms. of daboia venom, of 17 rabbits after ligature 7 survived, and the average death period of the remaining 10 was 86 hours; of 15 unligatured controls at similar doses there were only 2 survivors, and an average death period of 46 hours among the 13 fatal cases.

In fact, when ligature is applied after viper-bite, the action of the venom in causing local thrombosis comes into prominence, and, if sufficiently marked, this alone may result in no venom being absorbed, and the saving of life. The photograph on the screen shows the result, however, in a case of Russell's viper-bite : saving of life, but gangrene of the limb.

4. A piece of rubber tubing tightly applied gave better experimental results than a cotton bandage or a handkerchief or pugaree. Compression of the bitten limb by an Esmarch bandage did not give better results than simple ligature.

5. To be of any value in cases of colubrine-bite amputation must be immediate. Our main experiment on this point is shown on the

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screen. The M.L.D. of cobra venom for the monkey, *M. rhesus*, is 2·4 mgms. To each of ten such monkeys, under ether-chloroform anæsthesia, 24 mgms. or 10 M.L.D.'s were given hypodermically into the tip of the tail. The tails were then amputated three inches higher up at intervals of 1, 2, 3, 4, 5, 6, 7, 8. 9 and 10 minutes respectively. The results were as shown. The monkey with amputation at 8 minutes died. Those with amputation at 7, 9 and 10 minutes survived, but only after severe symptoms of venom intoxication. We may conclude that when 10 M.L.D.'s have been given, 1 M.L.D. may be absorbed into the circulation within 7 minutes. In the case of man where a cobra has given 211 mgms., or about 14 M.L.D.'s, one or more M.L.D.'s will be absorbed into the circulation within about 15 minutes of the bite. Amputation within this quarter of an hour interval may save life, and often will do so. Amputation at a later period will be useless.

6. In viperine-bites, where there is a tendency to local thrombosis, immediate amputation will frequently save life. Its especial rôle is in cases of bite from the Russell's viper. In the case of bites by the Indian species of the genera Lachesis and Ancistrodon the risk to life is so little that amputation is not justified.

7. In the case of colubrine-bites artificial respiration will become a useful measure, if some antidote can be found which will neutralise the venom after its absorption into the circulation.

8. Incision into the site of the bite and forcible expression of the venom are measures of little if any use. Cauterisation with heat is less effective than is cauterisation with chemical agencies. Excision of the bitten area is a measure analogous to amputation, and the same reasoning applies to this measure. Electrolysis, bleeding plus saline perfusion, and saline perfusion of the bitten limb are measures for whose value there is no experimental ground of evidence.

In the third section of our work we investigated the *value of chemical remedies in snake-bite*. Here the ground covered was very large. We tested experimentally some 200 chemical reagents in all, each selected as being of possible value. The main lines of experiment were analogous to those which went before. The *in vitro* experiments followed the customary method of mixing venom and reagent in a test-tube in solution, and later injecting the mixture into rats. But, thanks to our experimental curves, we could estimate what neutralisation had occurred, even in cases

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where the reagent had only partially neutralised the dose of venom, and where death still occurred. A glance at the cobra-venom curve will show this. Suppose we have mixed doses, each of 10 mgms., of cobra venom with a reagent, and have then injected these doses into rats of standard weight. Suppose the resultant average death period of these rats to be 11—instead of 69—minutes. This corresponds on the curve to a dose of only 2 mgms. of venom; and, roughly, we may say that the effect of the reagent has been to neutralise 8 mgms. of the venom. Similarly, in the *in vivo* experiments, the hypodermic injection of a measured dose of venom was followed by the hypodermic (or, in some cases, intravenous) administration of the remedy; and, even where death still ensued, we were able to estimate roughly the amount of effect which the remedy had exerted. Such "neutralising equivalents" cannot in any way be taken as accurate measures of the *actual* neutralisation which had occurred; but they will serve to test the *relative* values of one reagent against another. Throughout our work we relied first on the accumulation of very numerous observations on rats; following these up, where necessary, with more crucial experiments on small numbers of the more usual laboratory animals.

The reagents which neutralise venoms *in vitro* are very numerous. Apart from such physical reagents as heat, we may classify them as—

First, certain tissue extracts, such as bile; but the action of bile, at least in delaying death, seems solely due to its action in rendering absorption slower.

Secondly, many ferments: but their action is very slow, and often weak.

Thirdly, almost all caustics: and their action seems to depend solely on their power of causing proteid coagulation.

Fourthly, most oxidising agents, such as the chlorates, permanganates hydrogen peroxide, etc.

Fifthly, most of the better known antiseptics have a weakly anti-venomous action.

Sixthly, and by far the most important group: the soluble salts of the heavy metals, such as the chlorides or other soluble salts of gold, platinum, palladium, mercury, zinc, silver, etc.

It would be impossible to detail the experiments undertaken in this

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section of our work, except in a large paper, but our conclusions were as follows:—

1. The value of these local remedies is strictly limited by two factors: the necessity of injecting them before a fatal dose of venom has been absorbed into the circulation; and, secondly, the fact that only such venom as they happen to come into contact with on injection *in situ* will be destroyed.

2. They must therefore, be given before one M.L.D. of venom has been absorbed into the circulation. For man, for 10 M.L.D.'s. of cobra venom, this period is about 15 minutes. The more concentrated and the larger the dose of venom given, the shorter the period available within which such remedies will destroy all the venom injected.

3. The use of an efficient ligature increases this time limit.

4. Subcutaneous injection of these remedies in solution gives much better results than does incision and the rubbing in of moistened crystals. Under chloroform-ether anæsthesia twelve monkeys were given 10 M.L D.'s. of cobra venom hypodermically, and this was followed by the Lauder Brunton lancet technique: incision into the site of the bite and the very thorough rubbing in of crystals of potassium permanganate. The results are shown on the screen; all animals died. Experimentally, both in our hands and in the experiments of Surgeon-General BANNERMAN, the Lauder Brunton snake-bite lancet failed to show any real value. When one considers the facts of a case of cobra-bite: the injection of 14 or 15 M.L.D.'s. of venom; the 10 to 15 minute period within which one of these 14 M.L.D.'s. will certainly be absorbed into the system; the viscid character of fresh venom, and the difficulty of expressing it through an incision; the curved shape of the fangs and the fact that the venom has not been lodged in the subcutaneous tissues at the site immediately under the puncture marks on the skin—these completely disappointing results with the Lauder Brunton lancet are explained.

5. The subcutaneous injection of permanganate *solutions* gave better results than the Lauder Brunton technique. Here no incision is made; but the effort is made, by forcible injection into the site of the bite of a strong solution, to surround and destroy the venom injected before one M.L.D. can be absorbed. In this, as in all our experimental work, the experiments with monkeys and other larger laboratory animals were merely confirmatory, crucial experiments designed to test the

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results of much larger experiments, both *in vitro* and *in vivo*, on rats. The venom doses given were much larger than in the previous experiment: from 20 to 100 mgms. of cobra venom. It will be seen that, at such doses, the immediate injection of 10 c.c. of a 5 per cent. potassium permanganate solution saved 1 animal out of 5; 10 c.c. of a 5 per cent. zinc permanganate solution saved 3 out of 5; and 10 c.c. of a 5 per cent. calcium permanganate solution saved 2 out of 5. The salt of the heavy metal zinc was much better than the potassium salt.

6. Iodine trichloride and other well-known remedies gave less satisfactory results. Finally, we found the immediate subcutaneous injection of from 10 to 20 c.c. of a 1 to 5 per cent. solution of gold chloride to be by far the most efficient of these local remedies. The table on the screen shows the monkey experiment with cobra venom. At doses of

TABLE I.

Neutralisation of cobra venom by local injections of a 5 per cent. solution of gold chloride; Monkeys.

Monkey No.	Weight, gms.	Venom dose given.	Solution given locally.	Solution given.	Result.	Neutralising equivalent.
1	3450	20 mgms.	10 c.c. of 5 % gold chloride.	Immediately.	Survived.	1 c.c. = 2·0
2	2930	40 ,,	,,	,,	,,	1 c.c. = 4·0
3	3520	60 ,,	,,	,,	,,	1 c.c. = 6·0
4	4440	80 ,,	,,	,,	Slight sympts., survived.	1 c.c. = 8·0
5	3450	100 ,,	,,	,,	Died, 106 mins.	1 c.c. = 9·1
6	4550	20 ,,	,,	After 3 mins.	Survived.	1 c.c. = 2·0
7	3040	40 ,,	,,	,,	,,	1 c.c. = 4·0
8	3780	60 ,,	,,	,,	Severe sympts., survived.	1 c.c. = 5·8
9	2450	80 ,,	,,	,,	Died, 56 mins.	1 c.c. = 6·5
10	2010	100 ,,	,,	,,	Died, 49 mins.	1 c.c. = 8·2

Notes.—Venom solution, 1 c.c. = 100 mgms.

Control values on toxicity curve for the monkey: 20 mgms. = 43 mins.; 40 mgms. = 24 mins.; 60 mgms. = 18 mins.; 80 mgms. = 15 mins.; 100 mgms. = 14 mins.

Results.—Nos. 1 to 5: average weight, 3554 gms. 50 c.c. of 5 per cent. gold chloride given at once neutralised 291 mgms. 1 c.c. = 5·8 mgms.

Nos. 6 to 10: average weight, 3166 gms. 50 c.c. of 5 per cent. gold chloride given after 3 mins. neutralised 265 mgms. 1 c.c. = 5·3 mgms.

from 20 to 100 mgms. of cobra venom the gold chloride injections saved 4 out of 5 animals if given immediately, and 3 out of 5 if a delay of three

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minutes had occurred. With Russell's viper venom at similar doses 5 c.c. of a 1 per cent. solution of gold chloride saved 3 out of 5 animals; and 4 out of 5 animals in the case of doses of from 5 to 25 mgms. of Echis venom. Experimentally we found gold chloride to give better results than any other substance—except one, to be mentioned later.

TABLE II.

Neutralisation of Daboia and Echis venoms by 5 c.c. of a 1 per cent. solution of gold chloride given locally at once; Monkeys.

Monkey No.	Weight gms.	Venom and dose given.	Gold chloride solution given.	Results
1	2550	2 mgms. Daboia venom	5 c.c. at once	Survived.
2	2620	40 ,, ,, ,,	,, ,,	,,
3	4800	60 ,, ,, ,,	,, ,,	,,
4	2780	80 ,, ,, ,,	,, ,,	Died, 10 hours.
5	3800	100 ,, ,, ,,	,, ,,	Died, 15 hours.
6	2400	5 ,, Echis ,,	,, ,,	Survived.
7	2820	10 ,, ,, ,,	,, ,,	,,
8	2300	15 ,, ,, ,,	,, ,,	,,
9	4330	20 ,, ,, ,,	,, ,,	Died, 24 hours.
10	4400	25 ,, ,, ,,	,, ,,	Survived.
11	2770	10 ,, Daboia ,,	*Nil.* Control.	Died, 5½ hours.
12	1790	5 ,, Echis ,,	,, ,,	Died, 8½ hours.
13	1650	10 ,, ,, ,,	,, ,,	Died, 3½ hours.

Note.—Daboia venom solution, 1 c.c. = 100 mgms.
Echis venom solution, 1 c.c. = 25 mgms.

7. Further, from *in vitro* experiments, we found that these remedies exert a bigger neutralising effect when brought in contact with large doses of venom in concentrated solution than when acting on small doses of venom in dilute solution.

8. Intravenous injection of such remedies is useless, and the remedies themselves alone may cause intravascular clotting, pulmonary embolism, and death. I mention this, because one authority once suggested the use of 1 in 1000 solution of potassium permanganate intravenously as a line of treatment for snake-bite. Experimentally, in rabbits, we found that this remedy might cause death *per se* in the absence of any venom injection.

9. These remedies form the first line of treatment in a case of snake-bite seen at once. In the case of bites from the poisonous colubrines, however, such local remedies are only of value if—

(*a*) An efficient ligature has been first applied.

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(*b*) If they are given before one M.L.D. of venom has had time to be absorbed into the circulation, *i.e.*, within ten to fifteen minutes or so of the bite.

(*c*) If the subcutaneous injection be so planned and given as to isolate the injected dose of venom within the tissues and get at and destroy it all.

Hence, in a case of snake-bite seen some hours after its occurrence the value of such remedies is very doubtful.

10. From all that has gone before you will readily understand that the value of these local remedies is far greater in cases of viper-bite than in cases of colubrine-bite; the time limit is longer; the venom itself possesses a tendency to cause local thrombosis, and local remedies in viper-bite have a far greater chance of success than in colubrine-bite. Our experiments confirmed this *pari passu*.

11. The action of these local remedies is simply that of causing local coagulation necrosis. They destroy both tissues and venom indiscriminately, and will save life only if they happen to reach all the venom injected. Localised gangrene is always liable to follow their use.

12. Once fifteen minutes have elapsed since the bite, and once one M.L.D. of venom is in the circulation the use of local remedies must be supplemented by that of systemic ones.

Hence in the *fourth section of our work* we attempted to make a very thorough study of antivenene, and of the nature of the venom-antivenom complex.

We owe antivenene to CALMETTE, and the world can never be half grateful enough to him for its introduction. In India the antivenene used is all prepared at Kasauli by immunising horses against the venoms of the cobra and the Russell's viper. It is a strictly specific agent, and has no power, on experimental tests, of neutralising other colubrine or other viperine venoms. I have recently read an account of a case of Echis-bite where recovery ensued after the hypodermic administration of 10 c.c. of antivenene. I have every experimental reason for thinking that the adminisiration of the antivenene in this case had no effect whatever on the result; and it is to be noted that the chances of escape in even untreated cases of Echis-bite may amount to as much as 50 per cent. For many years it was one of the late Major LAMB'S ideals to produce a polyvalent antivenene; but, as yet, no such antivenene is on the market.

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CALMETTE got two cobras brought over from Egypt to Lille. These snakes were in captivity for 102 days, and I may remind you how rapidly the yield of venom falls off in a snake in captivity, as experimentally determined in the first section of our work. On eleven occasions he made them bite on to watch-glasses; and, from all that has been already said, you will see how almost impossible it is for a cobra under such unnatural conditions to give a really full dose of venom. CALMETTE concluded that a cobra does not give more than the equivalent of about 20 mgms. of desiccated venom at a good bite. As we have seen this conclusion was entirely erroneous. A cobra can give anything from 0 to 570 mgms. at a bite, and about 211 mgms. is a mean dose.

Secondly, argued CALMETTE, 1 c.c. of antivenene *in vitro* neutralises 1 mgm. of desiccated cobra venom. Take the case of cobra-bite in man. The cobra has given about 20 mgms. of venom. The man can withstand about 14 without dying. Only 6 remain to be neutralised. A hypodermic injection of from 10 to 20 c.c. of antivenene will therefore be sufficient to cure all cases.

You will see that almost every assumption in this train of reasoning is wrong. The most important error of all is the assumption that the *in vivo* neutralising value of antivenene will about equal its *in vitro* neutralising value in the test-tube.

Taking a sample of standard Indian antivenene, of which we found experimentally that 1 c.c. neutralised 1 mgm. of cobra venom *in vitro*, we gave a series of rats each 1 mgm. of cobra venom—a dose which kills the rat in 147 minutes—and then attempted to save their lives by immediate hypodermic injections of antivenene. Saving of life occurred at 10 c.c., or, exceptionally, in a few cases at 8 c.c. In other words, whereas the *in vitro* value of this antivenene was 1 c.c. 1 mgm., its *in vivo* value was 1 c.c. equals only one-tenth of a mgm.. Hence even if CALMETTE'S assumption about the dose of venom given being 20 mgms. is right, his estimate for the amount of antivenene needed to save life is ten times too small. But if the real facts be considered, then we have the average bite as 211 mgms., the sub-M.L.D, for man as about 14 mgms., leaving 197 mgms. to be neutralised, and this requires the hypodermic administration of nearly two litres of antivenene. But fresh antivenene soon falls below its original neutralising value on keeping, whilst a really large cobra may give as much as half a gramme of venom at a severe bite. In

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brief, antivenene is a weak product, and very far from possessing the potency usually attributed to it. The table shows the volumes needed to save life even when given intravenously in man.

But there are two methods of improving these results. The first is by altering the mode of administration of antivenene. We gave eleven experimental monkeys a dose of 4 mgms. of cobra venom each; and as the monkey will just stand 2·2 mgms. without dying, this left 1·8 mgms. to be neutralised. We then compared the value of subsequent antivenene administration by different methods, and the results are shown on the screen. To get saving of life after severe symptoms 20 c.c. were needed hypodermically, 10 c.c. intraperitoneally, and 5 c.c. intravenously. The sample of antivenene used in this experiment was of *in vitro* value, 1 c.c., equals only 0.65 mgm., which shows how different samples vary. But you will see that intraperitoneal administration is twice as efficient as hypodermic use, and intravenous administration is nearly twice as efficient again. Note in particular monkey No. 9 of the series: survival with only slight symptoms after 4 mgms. of cobra venom and only 5 c.c. of antivenene intravenously. Here the 5 c.c. of antivenene intravenously neutralised 1·8 mgms. of cobra venom, or probably even more, since the symptoms were so slight.

The second possibility is that of obtaining a concentrated antivenene. Off and on we were at work on this particular subject for nearly two years, trying different methods of immunising the antivenene animals, both for colubrine and for viperine venoms, and testing different methods of serum concentration. At last we obtained an antivenene by using methods analogous to those for the concentration of antidiphtheritic serum—12½ times as potent as the original sample from which it was made. The chart on the screen shows the amounts of normal and of this concentrated antivenene respectively needed to save life in the monkey when given intraperitoneally after different doses of cobra venom. The difficulty with such methods of concentration, however, is that of keeping the final product aseptic, and the processes are very laborious.

Now there was one point about our antivenene experiments which was very striking throughout when cobra venom was being used. Where after an injection of cobra venom in a monkey sufficient antivenene has subsequently been given to save life, there follows a curious train of events. The symptoms of general bodily paralysis set in almost as soon

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as in an animal which has had no antivenene: the ptosis, falling of the head, staggering and final complete bodily paralysis. Finally, the monkey is lying quite unconscious and apparently fully paralysed in its cage. But there is no onset of the expected asphyxial convulsions. Instead the breathing remains very slow, very deep, but very regular. Hour after hour passes in this condition. Next morning, however, the monkey is found to be perfectly fit. There is no residual paresis even of any limb. The meaning of this is clear, and in a set of experiments with incubated mixtures of normal brain emulsion and cobra venom we found confirmation. Although cobra and colubrine venoms are absorbed with lightning-like rapidity from their site of injection in the subcutaneous tissues, yet their fixation in the cerebral tissues is only a slow and gradual process, only gradually deepening to such a degree that death is inevitable. Hence if any remedy can be found which will act on the circulating venom, as does antivenene, only more efficient in action, its chances of saving life will be considerable, even if administered at a late stage. In the case of viperine venoms we used incubated mixtures of lung tissue (which is full of capillary endothelium) and Russell's viper venom. Here we found the exact reverse. Owing to the tendency to local thrombosis, viperine venoms are but slowly and very irregularly absorbed into the circulation, yet their fixation in the cells of the capillary endothelium is a very rapid process. Once again we get a complete contrast between the two classes of venoms.

A further result follows from this, and this again we confirmed experimentally in two sets of experiments. After a given dose of cobra venom the administration of the dose of antivenene needed to save life is equally efficient if given in divided doses at half-hourly intervals. After viperine venoms the dose needed to save life must be given at once, and not in divided doses.

The consideration of such results brought us to a whole series of experiments, on the nature of the venom-antivenom complex. We have seen that the toxic elements in venoms are not of a simple character. The antivenomous principle in antivenene, however, is. The chart on the screen shows the thermal death-point of antivenene, and should be compared with the corresponding charts for venoms previously given. You will see that at 65° C. when the proteids coagulate, the antivenene loses all its antivenomous power. Chemical analysis of antivenene

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confirmed this also; and one may say definitely that the antivenomous principle is either a globulin, or else is precipitated from watery solutions by exactly those chemical and physical agencies which precipitate globulins.

In brief, we regard the venom-antivenom complex as being essentially of the nature of a precipitin phenomenon, and antivenene as an antiserum produced in the immunised horse by the injection of the foreign proteids of venoms. Further, the combination of venom-antivenom follows the laws of incomplete or reversible mass action. The union of venom and antivenom is firmest at a certain concentration of both; on either side of this fixed point the union is less firm. When acting on a constant dose of venom the first c.c. of antivenene neutralises so much; the second c.c. neutralises less; the third c.c. less again, and so on, until the curve becomes flat. On the other hand, when a constant dose of antivenene acts on increasing doses of venom it seems to neutralise some portion of all the venom present, however large the dose. The union is not a simple chemical one like that of acid and alkali. but forms an example of mass action.

I need not quote the conclusions of our work on antivenenes, as I have given as rapid a résumé of it as I could, but may pass to the final, concluding experiment of our work. We carried out many series of further experiments which still remain unpublished. We worked, for example, for several months on the question of the immunity of the cobra to its own venom. I have often seen one cobra kill another. The two snakes rear up their hoods; they make rapid passes at each other, as if armed with rapiers; finally, one gets a grip of the other, usually about the base of the hood. In an instant the two snakes are twined around one another and thrashing about the cage. Finally, one is killed, from shock. We succeeded in killing a cobra by intraperitoneal injections of cobra venom, with clinical symptoms of cobra venom intoxication and post-mortem findings to correspond. But it took three-quarters of a gramme of desiccated venom to do it—a dose which no cobra could give in nature; and when one cobra kills another in nature, death is due to multiple injuries and shock. We experimentally examined every tissue of the cobra in the hopes of finding some tissue extract with antivenomous properties, but found that the bile alone shows any such activity, and that cobra bile is in this respect inferior to ox bile. In brief, the cobra's immunity to its own venom seems due to a deficiency of the necessary complement.

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You will readily see from this outline of our work the treatment which we recommend for a case of snake-bite. It is as follows:—

1. Instantly apply a firm ligature. It should be applied at such a site that it will compress all the tissues against a single bone, and prevent leakage of venom into the circulation.

2. Secondly. if possible, kill and identify the snake. If it is a non-poisonous one, no treatment at all is necessary. If it is one of the small vipers of India, a dose of calcium lactate may be given, the ligature removed, and the patient watched. If it is one of the six really lethal Indian snakes, then further measures are necessary.

3. If it is certain that the snake was a lethal one, immediate amputation may be recommended, for a bite on finger or toe.

4. If the bite be elsewhere, or if it is only supposed that the snake was a lethal one, or if the case be seen more than fifteen minutes after the bite, dissolve a photographer's 15-grain tube of gold chloride in a minimum of water; fill a strong hypodermic syringe with it, and attempt by forcible subcutaneous injection to isolate and totally destroy the whole dose of venom injected. No incision should be made.

5. If the snake was probably a cobra or Russell's viper, give from 100 to 200 c.c. of antivenene intravenously. There can be little fear of giving too much. Our experimental antivenene control-rats stood up to 8 per cent. of their body-weight of antivenene, and monkeys up to 2 per cent. of their body-weight of antivenene intraperitoneally without any symptoms, and man will stand very large doses easily. If the injection has to be given hypodermically, the dose of antivenene must be three or four times as large.

6. At the end of twenty minutes or so the ligature will have to be removed. Now commence a most careful watch over the patient. Be prepared to go on giving more and more antivenene if symptoms of venom intoxication set in. Start artificial respiration if necessary. Give cardiac stimulants, and do not give up hope too soon. Remember that, in the case of colubrine bite, the character of the respiration is the clinical guide to prognosis, and that the onset of paresis of the body does not necessarily mean a fatal issue.

7. In the case of viper-bites be prepared for the onset of hæmorrhages hours or even days after the bite.

Our work was suddenly interrupted in August, 1914, as, with the

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onset of war, both Major ACTON and I were sent on service. We had time for just one final experiment, designed to test, as far as possible, under conditions as nearly as possible those in nature, the value of this advice. The details are given in Table III. Under anæsthesia nine monkeys each received 60 mgms. of desiccated cobra

TABLE III.

Monkey No.	Weight.	Dose of cobra venom inoculated.	Ligature put on after the injection.	Ligature applied for	Amount of 5 % gold chloride injected after ligature removed.	Amount of antivenene given.	Result.
1	3320	60 mgms.	3 mins. after.	20 mins.	10 c.c.	20 c.c. given half-hour after ligature removed.	Survived: very slight symptoms.
2	2820	"	"	"	"	"	"
3	3212	"	"	"	"	40 c.c. given half-hour after ligature removed.	"
4	2180	"	"	"	"	"	"
5	2520	"	"	"	"	60 c.c. after half-hour after ligature removed.	Survived; no symptoms.
6	3270	"	"	"	"	"	Died 80 mins.
7 control	2370	"	"	"	"	*Nil.*	Survived; very severe symptoms.
8 control	2020	"	"	"	*Nil* given.	*Nil.*	Died 57½ mins.
9 control	3200	"	*Nil.*	*Nil.*	*Nil.*	*Nil.*	Died 43½ mins.

Note.—Cobra venom solution used 1 c.c = 100 mgms.; dose given 60 mgms = 25 lethal doses for the monkey.

venom or twenty-five lethal doses. Three minutes later a rubber band was tied on the leg as a firm tourniquet above the site of injection, and was left on for twenty minutes. The ligature was then untied, and the site of the venom injection impregnated by a subcutaneous injection of 10 c.c. of 5 per cent. gold chloride solution. Still half an hour later, or 53 minutes after the bite, as it were, doses of from 20 to 60 c.c. of standard antivenene were given intraperitoneally. You will see the result in the last column of the table. No. 9 received the venom dose only and died in 43½ minutes. No. 8 was ligatured only, and died 57½ minutes after the injection. No. 7 received the gold chloride but no antivenene; this monkey survived after symptoms. Of the six animals which received the full

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dose and the full treatment, five survived, and none of them showed anything more than the slightest symptoms of venom intoxication. It ought to be possible to save at least 80 per cent. of all cases of bite from lethal snakes *if* one had these remedies at hand and used them sufficiently early.

But no one can pretend to be satisfied with this result. In the usual case of snake-bite in an Indian village, seen perhaps some hours after the bite, the chances would be far less. Major ACTON and I have not hitherto been afforded the chance of resuming our work on this subject; but it seems to me that the chances of finding a safe, efficient and reliable cure for the bite of even the poisonous colubrine snakes of India are good. Experimentally in the rat, we found one substance which was even better than gold chloride. The dichloride of palladium is a salt which is almost insoluble in water; but which yields a clear solution of about 1 in 2,000 strength. This solution, however, both *in vitro* and *in vivo* destroys venoms better than any other substance tested. Our experiments with this salt were confined to some forty-eight rats, and we have not published this finding because it has not yet been confirmed on larger animals. Now, in concentrating antidiphtheritic serum, there is one process in which the antitoxin is collected from watery solution by precipitating with a soluble zinc salt. It may be possible that a palladium dichloride solution, or some solution of some other heavy metal, such as gold, will precipitate the antivenomous principle from a watery globulin extract or solution of antivenene, and form a new compound of heavy metallic ion with antivenom suitable for solution, and possibly suitable for intravenous injection. Other substances which seem to warrant experimental trial are the colloid suspensions of the heavy metals. The problem, at least, has perhaps been clarified, and has become one for the biochemist.

DISCUSSION.

Sir LEONARD ROGERS, F.R.S.: I have listened to Major KNOWLES' paper with much interest, as, although I was investigating snake poisons seventeen to twenty years ago, I have never yet found time to read the very important paper of Major KNOWLES and Major ACTON. On the other hand, I have learned this evening from my friend, Major KNOWLES,

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whom I selected to carry on my work in the Tropical School at Calcutta, that he has overlooked a paper of mine on this subject, published in *The Philosophical Transactions* of the Royal Society in 1904. That paper deals with the physiological side of the subject, as I worked in Professor WALLER's laboratory in London University, on my first leave home from Calcutta, for six months in 1903, and made blood pressure and respiratory tracings of the physiological actions of different classes of venoms. It was first shown by Sir LAUDER BRUNTON and Sir JOSEPH FAYRER that colubrine cobra poison paralyses the respiration. The hæmolytic action was worked out in the cobra class by D. D. CUNNINGHAM, but it was very slight, because if you give a lethal dose, and examine the blood before and afterwards, it is found to have no effect on the blood. I worked out the action of the hamadryad and krait in paralysing respiration, and also an interesting one in the banded krait, which first produced a fall in blood pressure, which you do not get, as a rule, in the colubrine class, and subsequently there was respiratory failure, which caused death. I also worked at the sea-snake venoms, and they act almost exactly as the cobra venoms, and kill by means of paralysis of the respiratory centre, but they are, weight for weight, ten times more venomous than is the cobra venom. The two which were the most venomous were Enhydrina and the Platurus, though in one text-book the last-named is given as non-venomous. But the most interesting work was on the viperine class. It was shown by WEIR MITCHELL in America, and C. J. MARTIN in Australia, that some snakes might produce vasomotor paralysis, but the most important factor of the vipers was the hæmorrhagic action, which Major KNOWLES has told us about. In the Russell's viper of India, if you give a lethal dose to a small animal the essential feature is clotting in the vessels, the animal dying of convulsions in one to three minutes. I was taking blood-pressure tracings, and when I worked with the minimum lethal dose of Russell's viper venom I found that there was a rapid fall of blood pressure, and then the respirations nearly ceased, and we thought the animal was dead. Then respiratory convulsions occurred, and the pressure rose again. It then fell and rose again, oscillating three or four times, but when the animal died there was no trace of clotting in the vessels. In the case of a man who has been bitten by the Russell's viper, I have not seen any records in which clotting of the blood was found. You get the opposite effect in the

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way of loss of coagulability. If an animal dies in response to a smaller dose, you will still find intravascular clotting, but the rest of the blood is not coagulable, and that accounts for the hæmorrhages which occur at the later stages if those bitten happen to live for some time. I investigated this fall of blood pressure, and I found that every pulse-beat could be seen, showing that the heart was still working, while the way in which the pressure was pumped up with the respiration convulsions suggested it might be a vasomotor paralysis which was taking place. I questioned whether it might be a clotting of pulmonary capillaries, but I excluded that by transfusing unclotted blood, and I found it ran through with a low pressure. I then queried whether it was a vasomotor paralysis. I found that if you injected Russell's viper venom it was possible to produce only a fall of blood pressure (which you could do by giving a minimum lethal dose first, which enables you to give any dose without producing intravascular clotting). If you first cut the spinal cord and then injected the venom, there was no fall of blood pressure, while if a dose was first introduced and then the cord cut there was no further fall of pressure. I next put the mesentery under the microscope and then injected the serum, and as the tracing was being taken and the blood pressure was falling, I saw the blood-vessels dilate in the portal system. I then put the intestine in the oncometer and so registered the blood pressure and the volume of blood in the intestine, and the latter rose while the former fell. Thus I demonstrated that the essential action in man was vasomotor paralysis without any intravascular clotting. Professor STEPHENS gave me some puff-adder venom, but it had much less clotting power than that from the Russell's viper. There was a great excursion of the blood-pressure lever, showing vaso-dilatation of the blood-vessels. It was the same with rattlesnake poison, which was important, because rattlesnake venom does not cause clotting, except rarely, just at the site of injection. Thus it was proved that the essential lethal factor of the viperine snakes as a class was a vasomotor paralysis. Then I tested with adrenalin, and I found that if you use a viperine venom, such as that of the rattlesnake, and give a dose which does not prove rapidly fatal, you get a marked fall of pressure, but on then injecting a dose of adrenalin the pressure ran up again to above the normal, and remained high for a long time, showing that that substance had a definite counteraction against these venoms, and that is why

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I suggested using that drug and pituitrin in viperine poisoning. In the case of the banded krait, there was, first of all, a fall of pressure, then a rise, then respiratory paralysis. That was due to a mixture of colubrine and viperine toxins, the main toxin being the one which acted on the respiratory centre. Thus colubrine venoms act essentially on the respiratory centre and viperine venoms on the vasomotor centre, and so I suggested we should be able to get an antivenene against vipers as well as against colubrines, which was done later in India by LAMB. But I also showed that the amount of antivenene required to be used is too great to make it of real practical value in most cases. CUNNINGHAM obtained 250 mgms. of venom for a full cobra bite, and in the case of the king cobra I got 800 to 900 mgms., so that the king cobra bite required over 1,000 c.c. of an antivenene serum to neutralise it, which must be given very quickly and intravenously to be of use. Antivenene against cobra has more effect, but also requires large quantities. I have always urged that it is best to first use local treatment, so as to neutralise as much of the venom locally as possible, as, if you reduce the dose in the body from several to about two minimal lethal doses, you have then a good chance of benefit from the antivenene, but you must give it intravenously. LAUDER BRUNTON'S suggestion was that his snake lancet should be carried about in the waistcoat pocket, as it contained a supply of the required permanganate. The only case of snake poison I saw was in Calcutta, in the European General Hospital Compound, when a man ran in, saying a servant had been bitten by a cobra. I had my lancet ready and I treated the patient at once, and he recovered. Of course, we cannot say if he received a lethal dose of venom. Out of twelve cases I published, in which the snake was killed and identified as poisonous, we obtained ten recoveries, though one was not treated until several hours afterwards. I agree it is only occasionally that one gets a case in time, while antivenene is rarely available.

With regard to my experiments, Major KNOWLES has referred to the unsuccessful use of LAUDER BRUNTON'S method in the monkey. I had a licence for only twenty experiments, and worked with rabbits and cats, but a few rabbits died quickly, so I went on with cats, and I saved several of them after injecting several times a lethal dose. The measure of success varies with the particular animal's tissues, and the cat is more resistant than the rabbit or the monkey. Antivenene should be used as well when it is available.

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I have been interested in what was said about gold chloride. In 1895 CUNNINGHAM was testing gold chloride, and he found that gold chloride was more effective than permanganate or other drugs for injection. But the difficulty in these cases is that you so seldom have a syringe and solution ready in time, so the lancet containing permanganate is a more practical method. But there is no reason why we should not go on experimenting as Major KNOWLES has, and, I hope, before long he will be able to produce an even better method than we have yet secured.

Major E. BLACK: I wish to refer to one thing in this which is absolutely the best paper on the subject I have ever come across. I feel justified in saying that, because many years ago I conducted similar experiments, and I may say I have been bitten by a snake too. We in this Society wish to guide those inexperienced in such subjects. I have had the misfortune to discover, in many countries, that there is a widespread delusion to the effect that the antivenene of CALMETTE should be used in all cases. My experience many years ago not only proved the point made in this paper that it is quite futile in relation to other species of snakes, but the matter was worse than that. In all my long series of animals bitten by other species of snakes in my experiments, those which had CALMETTE'S antivenene died more quickly than did the controls. It should be widely known that antivenene, to be of value, must be prepared in relation to the particular species of snake whose bite you are treating.

Major H. C. BROWN: I had the good luck to be in Kasauli when Major KNOWLES and Major ACTON were conducting their experiments and I would like to congratulate them on their work.

The last speaker referred to the futility of treating snake-bites of one species with an antivenene prepared for a different species, but in the case of viperine bites, I consider that the possibility of controlling the hæmorrhages by means of a non-specific antivenene is well worthy of attention and further work.

I have had a certain amount of clinical experience of the treatment of epistaxis and other similar cases of hæmorrhage with normal horse serum and am of opinion that the results obtained were satisfactory. It was in view of this that in the case of a patient who was sent to me two

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days after he had been bitten by an *Echis carinatus*, I tried the effect of a subcutaneous injection of normal horse serum. The patient exhibited extensive hæmorrhages which appeared rapidly to respond to the normal horse serum. Although this is an isolated case, I think the treatment is worthy of further investigation and should like to ask Major KNOWLES whether he has tried this or non-specific antivenenes in viperine bites.

I think that Major KNOWLES has, if anything, underestimated the importance of his work on the concentration of antivenene, for not only should this be of extreme value in the treatment of snake-bite, but also it is of great importance in its relation to the immunization of horses.

Those who have had experience in this know that the abscess formation is very troublesome, and if horses could be kept on small doses of approximately 50 mgms. and the antivenene subsequently concentrated 12·5 times or more, much of the abscess formation could be obviated.

I should like to ask Major KNOWLES whether the antihæmolysins in antivenene are correlated with the antineurotoxins.

Major KNOWLES: I have to apologise for the length of my paper. I concentrated as much as possible on the actual findings. I thank all who have taken part in the discussion, and especially Sir LEONARD ROGERS. With regard to the Lauder Brunton lancet the paper was somewhat dogmatic because of the need for cutting down one's remarks. One admits the portability of it, and that it neutralises some venom, but the results are not comparable with those from the injection of permanganate solutions.

With regard to gold chloride, Major ACTON found mention of it in CALMETTE'S book.

With reference to the specificity of antivenene, and the point that Major BROWN makes as to using antivenene in Echis bite, what suggests itself is that it was simply a hæmostatic action on the part of the horse serum. I remember Major BROWN at Kasauli remarking that, if our antivenene results were to be published, a regiment of horses would be required to produce the amounts of antivenene which would be indented for. If the immunising dose for horses be kept down to 50 mgms. it is

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easier to concentrate a weak antivenene than a stronger one and the results with such a concentrated antivenene might be better. With regard to antivenene losing its power when the proteids coagulate I referred only to its antineurotoxin.

The PRESIDENT: We have had a very interesting paper full of new facts and observations. I spent three hours with CALMETTE in his laboratory in Saigon in 1900, and I was brought up in the CALMETTE school. I have dropped the subject since, because I thought I knew all about it; but I find that much of what I learned from CALMETTE has been, to a large extent, superseded. Knowledge, however, has accumulated of late years on snake-bite, and we are much obliged to Major KNOWLES for placing it before us. I know something about snakes in China, and I have had not a few escapes. Is it true that when a native is bitten by a snake in India, he chases the snake for some distance to find out whether it is a poisonous one, before he does anything to treat the bite? The custom is different amongst the Chinese, for, if one of your chair coolies is bitten by a snake in the foot, they drop you at once, take off the pole cloth they carry over their shoulders, and with the aid of the poles they carry, make a tourniquet, which is applied above the seat of the bite within one minute after the bite. I do not know whether Indians attempt anything of the kind, but the instinct of the Chinese is to do the right and practical thing on the instant; so that deaths from snake-bite in China are rare. The second case I had to treat in a Chinese hospital was that of a man who came in with a circle of his skin, just above the wrist, taken clean away; there was another circular wound just below the elbow, another above the elbow, and another immediately below the shoulder. I could not even guess the cause of the condition until I ascertained that he had been bitten by a snake. He had applied four tourniquets, and kept them on so long that he had gangrene at three spots; it looked as if the skin had been carefully dissected off. There had been efforts at healing and he remained in much the same condition six months after the occurrence.

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EXTRACTS FROM THE TROPICAL DISEASES BULLETIN.

McKINNON (MURDOCH). **European Children in the Tropical Highlands.**—*Lancet.* 1920. Nov. 6. pp. 944-945.

The author, Consulting Physician to the European schools, Nairobi, records the conditions found on examination of the hearts of 200 children resident in British East Africa: all were either born in the country or resident there 2-10 years. Nairobi has an altitude of 6,000 feet, is three degrees south of the equator, and possesses, according to the author, the finest climate in the world. He considers that only by the study of comparative statistics like these can a conclusion be arrived at as to the suitability of a given tropical country for permanent European settlement. It appeared to him that breathlessness and lassitude are two of the most noticeable changes occurring in European children of school age. The children were from 7 to 18 years of age, and the observations, taken at rest and after exercise, included the pulse-rate, respiration-rate, area of cardiac dullness, and blood pressure. The exercise consisted of a run of 50 yards in a circle. The average pulse-rate at rest in children of 7-10 was 93 per minute; and from 10 to 15 it was 86. [The number of children at the respective ages is not stated.] After exercise there was an increase of rate of 37 beats per minute. The return to the normal after exercise took on an average 22 minutes, a much longer time than has been recorded in healthy hearts at lower altitudes in temperate regions. After exercise 12 per cent. of the cases showed some irregularity, usually a missed beat with an apparent slowing of the pulse, but after the age of 14 no irregularity was noticed. Definite dilatation of the heart, determined by auscultation, was present in 6 per cent. Only one child born in the country was suspected of having an organic lesion, and two others in whom there was a murmur came from South Africa. "Rheumatic fever and chorea . . . are very rarely seen among children in tropical Africa." The author stresses the importance of the delay in return to the normal pulse-rate, as indicating the amount and nature of exercise permissible.

A. G. B.

OSBORNE (W. A.). **The Physiological Factors in the Development of an Australian Race.** *Med. Jl. Australia.* 1920. Sept. 18. No. 12. pp. 261-267.

This was a popular lecture given by the Professor of Physiology, University of Melbourne, at the Australian Medical Congress in August, 1920; it contains much that is of interest.

The juxtaposition of Australian and British troops in Egypt, France and Britain drove into the minds of onlookers the fact that there were physical differences, that an Australian race is in process of formation, and this is apt to be ascribed to climate. But, the author writes, "it is impossible to point to any experience of climatic action uncomplicated by other and very potent factors." Such are race, food, disease, form of government, economical conditions, These factors are discussed in the case of Australia.

Under race is noted the great advantage, possessed by Australia, of political unity. As to food, "in man's history food may be claimed to have been of immensely greater importance than climate." Extracts are given of the Lenten fare of a Northumberland household in the 16th century, to show what mighty feeders the English were. [This was an aristocratic family. What of the labourer?] The England of the 20th century is underfed. "The Australian," Professor Osborne writes, "is the greatest meat-eater in the world. . . . Pure, varied and abundant, Australian food must be given a big share of the credit for the physique of the Australian and for some part of his initiative and courage." As to the future, cereals will increase and beef animals decrease, but there are immense possibilities in pig-raising and the fisheries are almost untouched. For diseases, with the exception of hookworm and

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dengue. Australia enjoys a singular immunity from the worst tropical diseases, and it has the advantages of the absence of a large native population and of slums, except in a few large cities. A tribute is paid to the quarantine department, which has kept out plague and other infections.

Lastly, he comes to climate and the effect of tropical conditions on the white man. He pictures a highly developed civilisation in Central America, and the question being raised whether North America is habitable by men. A Royal Commission investigates and reports that this region is either uninhabitable or, if habitable, would be peopled by degenerates. To retain the body heat artificial coverings would have to be worn, which would prevent the skin being kept in a clean and healthy state; the people would have to live in enclosed rooms warmed artificially, and the purity of the air would present an almost insoluble problem, and so forth; a flight of fancy to show the importance of adequately allowing for man's adaptability.

Two disabilities can be definitely ascribed to tropical climates: (1) to keep the temperature normal the skin must always have a liberal supply of blood, so that the emergency supply to other organs is not so great as in temperate climates; and (2) a fever temperature is very easily acquired in exertion, and this excess heat takes some time to escape, the result being that the combustions occurring in the body are quickened. Neither of these objections is considered to be serious. The author notes the relative immunity of Australia from heat-stroke. He is convinced that in India and Mesopotamia some disease like fulminating malaria is the cause: "the mere mention of dry skin preceding the fatal issue, is to me sufficient evidence to mark the condition as due to infection."

As to the favourable action of climate in developing the hardy races in the north of Europe, "the most furious assaults that Mediterranean civilisation ever encountered came from the scorching deserts of Asia and Africa." He suggests that houses in the tropics may in the future be made more habitable by cooling installations, and refers to the unsuitability of the buildings in tropical Australia.

"To sift out what is due to disease and what is due to climate is the problem which must be solved before we can speak dogmatically on the chances of the white man prospering in the tropics, and I venture to say this problem has not yet been properly attacked, largely through lack of evidence." A. G. B.

BALFOUR (ANDREW). **The Treatment of Smallpox by the External Application of Potassium Permanganate.**—*Jl. Trop. Med. and Hyg.* 1921. Feb. 15. Vol. 24. No. 4. p. 37.

The treatment of smallpox by the external application of potassium permanganate was introduced by DREYER, of Cairo, in 1910, and has been practised chiefly by the Germans. "When the patient is admitted to hospital his whole body is painted over with a freshly-prepared saturated solution (five per cent.) of potassium permanganate. On each successive day the same solution is applied, unless the skin is found too sensitive, in which case a weaker solution is employed, one of 1·5 per cent. being often suitable." The good results are probably to be attributed to the germicidal and oxidising powers of the drug. It appears to be of signal service in lessening the suppurative process. It is also said to prevent complications, the formation of bed sores and the occurrence of general sepsis. The pitting of the skin is reduced. It should be used, at any rate, in severe cases in the tropics, where facilities for proper nursing and attention are lacking, but practitioners should note that DREYER recommends caution in cases where the heart is diseased or feeble. A. G. B.

CHAPIN (HENRY DWIGHT). **An Epidemic of Kaffirpox in Jamaica. Random Notes of a Tourist.**—*Med. Record.* 1920. Oct. 2. Vol. 98. No. 14. pp. 553-554.

The epidemic began in May. In August the author visited an isolation hospital in the suburbs in Kingston, where 300 patients were seen. These cases differed clinically from both smallpox and chickenpox. The pocks were abundant on the face, and were scattered over the wrists, arms, body and legs. The palms of the hands and soles of the feet were covered with

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pocks. In one case vaccination had been done with success after the appearance of the eruption. Pocks were seen in a number of throats. The pustules were unilocular in the cases examined, and there did not appear to be a true umbilication. The pocks were fairly uniform in age. A deep purple staining of the skin persisted for several weeks after scabbing. The patients were only sick at the commencement, with malaise, headache and backache. "It was amusing to see many patients, with face and body covered with pustules, walking about the grounds, half-clad and smoking cigarettes as if nothing were the matter, although they had every appearance of smallpox." There were three fatal cases, but death, it was thought, was not directly due to Kaffirpox Infants and young children usually have the disease in a mild form.

The author quotes CASTELLANI and CHALMERS as to the history and geographical distribution of the disease, and its relations with smallpox and chickenpox. A. G. B.

GOLDSMITH (G. M.) AND LOUGHNAN (W. F. M.). **Notes on Three Cases of Alastrim.**—*Jl. Roy. Army Med. Corps.* 1921. Jan. Vol. 36. No. 1. pp. 66-68.

These notes refer to three cases admitted to hospital in Jamaica in May and July, 1920. The disease is said to differ from smallpox in that vaccination affords but slight protection, the severe lumbar pain of smallpox is absent, the secondary fever is very mild or is absent, true umbilication of the scabs is not seen, in adolescents the disease is very mild, scarring is absent, the mortality is not more than 1·5 to 2 per cent, It differs from varicella in that it occurs at all ages, the vesicles tend to become confluent, the stages are more prolonged. The authors say [contrary to CHAPIN, *see* above] that the rash is not seen on the palms or soles.

A. G. B.

MANN (W. L.), HELM (J. B.) AND BROWN (C. J.). **An Edema Disease in Haiti. A Preliminary Report.**—*Jl. Amer. Med. Assoc.* 1920. Nov. 20. Vol. 75. No. 21. pp. 1416-1418.

The authors are medical officers in the United States Navy, and the disease described was seen among prisoners, especially at Cape Haitien and Port au Prince. The authors write:—

"The clinical manifestations of this disease simulate beriberi, but no paralysis of the extremities has been noted, and the rate of the heart-beat is slower than in beriberi. It likewise resembles 'prison,' 'war' or 'nutritional' edema, but the Haitian dropsy is apparently more rapid in development, the mortality rate is greater, and a change in diet and environments has not been observed to have any marked effect as a curative. The symptoms and the incidence of the condition under discussion correspond somewhat closely to 'epidemic dropsy,' and may be differentiated from it by the absence of fever."

They saw 3,000 cases, and studied more than 200 necropsies.

"The edema disease is an acute or subacute condition of at present undertermined causation, characterized by the cardinal symptoms of edema, emaciation, weakness, diarrhœa, subnormal temperature and a slow pulse rate. These symptoms, one or more of which may be absent, exist in varying proportion. The dropsical condition may appear and disappear at irregular intervals."

It was stated that the disease had existed in Haiti for many years, and the informant gave a name by which it is designated. It is chiefly present in the prisons; in some it is common, in others, "under almost identical conditions as to food and climate," it is rare or absent. The disease was at first believed to be limited to prisoners interned for less than three months. Later, cases were noted among the long-term prisoners. "Edema has developed in several cases in less than fifteen days after incarceration." As to causation, all investigations and experiments [the details of which are not given] tend to negative the natural assumption that the diet was at fault. The etiology is still under investigation. There is no evidence of any relations to conditions existing prior to confinement. Thus, 40 bandits when captured were in normal physical condition, but 12 days later a case of œdema developed, and within seven weeks 25 per cent. were affected.

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The notes on symptomatology are based on an intensive study of 50 cases, and the symptoms are taken up in turn. The œdema usually begins on the dorsum of the feet, pits on on pressure, and may go on to general anasarca. Diarrhœa occurred in 28 of the cases. It is stated that the stool often consists of 5 or 10 c.c. of pus. Emaciation may be extreme, and it is weakness usually which brings the man to the sick bay. The average temperature is 97 to 97·5°; the average pulse rate 57 per minute. The duration in fatal cases is about 25 days. In the necropsy findings there is nothing characteristic, nor do the laboratory findings help to a diagnosis. A. G. B.

LUZ (FERNANDO) AND TORRES (OCTAVIO). [Translated from the Portuguese by MCSHANE (A.).]
Case of Gundu observed in Bahia, Brazil.—*New Orleans Med. and Surg. Jl.* 1920. Sept. Vol. 73. No. 3. pp. 100-104. With 4 figs.

The authors' patient was a mulatto, a native of Bahia, aged about 60, from whom no history could be obtained. The patient had "two tumours at the root of the nose; two swellings, each three centimetres in length and one in width, situated one on each side of the inferior maxilla, and one centimetre above the free border of the latter; exostosis of the following long bones: ulnas and radii; tibia and fibula, several ribs, the humeri, clavicle, metatarsal bones, and some metacarpal bones. . . . The feet turned outwards, making an angle of more than ninety degrees; the inner border of each foot did not follow a straight line, but each border formed an obtuse angle of 160 degrees outwards at the level of the tarso-metatarsal articulations."

Later the patient developed paralysis of the lower limbs and relaxation of the sphincters; he was transferred to the isolation department and died. It was not practicable to make a complete autopsy, but the head and both legs were removed and photographs were taken,

"The lesions were as follows: piriform tumours of the ascending apophyses of the superior maxillary bones, which encroached slightly on the lower border of the orbit and the nasal fossæ, gradually diminishing towards the malar bones; on the lower maxilla, there were two tumours three centimetres in length and one and half in width, lying parallel with the maxillary border. These tumours were of the same nature as those above described . . . .

"Small exostoses of the cranium, which were perceptible during life, were more evident after death.

"The tibias and fibulas, which presented large exostoses, easily seen in the photograph, were the seat of interesting lesions. The lesions are seen to be clear: the tibias have lost their anterior border entire in their length, and were almost doubled in thickness, the tumours being best developed in the lower two-thirds. There were close union between these bones and the fibulas, which were adherent at the upper and lower ends. The tibial *plateaux* showed exostoses laterally.

"The thickened fibulas seemed to be twisted on their long axis, being thickest in the lower two-thirds. In the upper third we see perfectly the union of these two bones with the tibias.

"Apparently the long lesions are those of osteo-porosis. . . . .

"We made longitudinal sections of one of the tibias, not only to see the shape of the bones, but also to study the medullary canal.

"The dimensions were visibly increased in all directions: the medullary canal was extremely reduced, presenting a small orifice, which was not sharply limited, but was lined by a kind of reticulated bony tissue, the deeper portions of which blanded with the cavity of the bone (rarefying osteitis).

"On the posterior surface of the tibia, a well-developed bony ridge is seen."

Sections of the bony tissue are being studied.

The photographs show the patient's profile in life, his skull, the leg bones and a transverse section of the tibia.

This seems to be the second case of goundou described from America, the first having been reported by MENDES in a mulatto in 1901. A. G. B.

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REVIEW.

DAUKES (S. H.), [O.B.E., M.B., D.P.H., D.T.M. & H., Wellcome Bureau of Scientific Research]. Barrier Charts for Health Officers. A Synopsis of Preventive Measures against Communicable Disease in Four Tables. Four Folded Charts in a Wallet, measuring 10ins. by 5ins. Published for Wellcome Bureau of Scientific Research by Baillière, Tindall & Cox, Henrietta Street, London. Price, 3s. 6d. net.

This publication consists of four folded charts on stout paper, in a green manilla cover closed by a stud: it contains also a page of introduction by Dr. ANDREW BALFOUR and an explanatory preface by the author. Dr. BALFOUR tells us that the Charts were designed by Dr. DAUKES when in charge of the School of Army Sanitation at Leeds. He believes that they will be useful to Medical Officers of Health and Sanitary Inspectors. Dr. DAUKES writes in explanation of his title, "Sanitation is the barrier which a civilised community places between the sick person or germ carrier and the healthy population." The Charts deal with four groups of infections: (1) the alimentary, (2) the inoculative, (3) the respiratory, (4) the contact group. The alimentary group is least happily named, for the adjective does not refer to the mode of entry of the infective agent, as it does in the other groups, but to its means of exit with the urine or fæces, as indicated in the sub-heading; thus it includes conditions—schistosomiasis and ankylostomiasis—in which infection is usually produced by skin contact with the young stages of the parasites: this, of course, is stated in the body of the Chart.

The information tabulated for each disease consists of the locale of the infective agent, incubation period, period of infectivity, observation period from latest period of infection, 1st barrier, results of defective 1st barrier, 2nd barrier, 3rd barrier, results of defective 2nd and 3rd barriers, 4th barrier, final result of defective barriers. The "barriers," which are printed in red, concern (1) the sick person and carriers, (2) the zoological factors, (3) general hygiene, (4) immunisation by vaccines, sera, drugs. The zoological barrier is blank in the respiratory group. Other gaps indicate, for the most part, gaps in our knowledge.

There can be no question of the value of these Charts, either in the pocket for handy reference or on the wall of the office, and, apart from their practical use to the hygienist, they are educative and suggestive.

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TRANSACTIONS

OF THE

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

OCTOBER 20TH, 1921.

VOLUME XV. No. 4.

Proceedings of a meeting of the Society held on Thursday, October 20th, at 8.15 p.m., at 11, Chandos Street, Cavendish Square, W. 1, Sir JAMES CANTLIE, K.B.E., F.R.C.S. *(President)*, in the Chair.

LIFE INSURANCE IN THE TROPICS.

Inaugural Address.

BY THE PRESIDENT, SIR JAMES CANTLIE, K.B.E., F.R.C.S.

I am very much obliged to you for the kindly reception you gave me the other night, at my installation as President, and again to-night.

The subject I have chosen for what is called my Presidential Address is one which I think interests every one in the room; you are every one interested in the tropics, whether medical man or layman. It is an absorbing question. Amongst us are medical officers of insurance companies, medical examiners for companies, banks and commercial firms, for members of their staff who are about to proceed to the tropics, and who examine their men on their return on leave.

I have interested myself in the matter for some twenty years, and published several articles and read several papers on the subject. As far back as 1897, a paper was published in the *British Medical Journal*; I addressed a meeting of the Life Assurance Medical Officers'

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Association in 1903; and, further, read a paper before the Royal Institute of Public Health at one of their Conferences.

The question before us seems to resolve itself into—Does residence in the tropics impair the health of the individual to such an extent as to justify the imposition of an extra premium being demanded by insurance companies. In insurance prospectuses I see that a dangerously warm country is regarded as one situated within 33° north of the equator and 30° south of the equator.

It is, of course, well known that within the area specified we meet with climates of widely different characteristics, varying between climates of an equatorial, a tropical, a sub-tropical and a temperate character; climates, some with an insular and some with a continental type of character. To slump these together, as some of the companies say they do, is perhaps an inevitable method of dealing with such a huge subject as tropical insurance, but it is hard on individuals proceeding to a "good climate" area within the specified longitude and latitude, and one wonders how the rules appertaining to the amount of extra premium demanded were drawn up. Was it from statistics of the percentages of deaths published by the local governments in Crown Colonies, etc., or was it from information received from travellers? If not, one cannot conceive what were the sources from which the knowledge was derived—say fifty years ago. I have described the tales we hear of these distant tropical lands as ship captains' gossip gathered from the common talk of those lands when the ship goes in harbour. On the way out in 1887, the officers of the ship—whether sailors or engineers—spoke of little else than the fate of the passengers whom they had taken out on previous voyages, how they were dead in twelve months or sent home wrecks after a few years. When again one's friends and relations at home heard one was to go to the eastern tropics, one was deluged with letters, cuttings from reports, callers beseeching one to give up the idea of going out to a death-trap, a "white man's grave," and so forth, until one wondered how any Briton ever went out at all.

No wonder some of the Insurance Companies of that date said they would have nothing to do with insuring the lives of persons going to the tropics on any consideration whatever. Were the extra premium charges founded on the local government reports, here, again, the information was faulty in a superlative degree. None of our tropical colonies

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produces its own labour; the white population is imported from Britain and the native labour from the neighbouring countries, or brought from overseas: Tamils, etc., to Ceylon, Chinese to the Malay States and to the Straits Settlements, Indians and Chinese to the West Indies, and so forth. They come as young adults as a rule without their wives and children; without, in fact, the part of the family which swell the death-rates in home countries. The white population consisted of bachelors for the most part, who, on being carefully examined, were sent out in perfect health; often taught by lectures how to avoid diseases; re-vaccinated against small-pox, inoculated against typhoid, and occupying dwellings—often of a better class than they were accustomed to at home.

When they get ill they are carefully attended to, sent for a change or sent home on sick leave. What are the diseases that give the district a bad name? Malaria is the chief, but malaria kills but a small percentage of those attacked. It is a disease that incapacitates, and the loser is the employer or the firm in which they work—not the insurance office, whether the insurance is for a period of years or for life.

Besides, malaria, its cause, prevention and treatment is so well known at the present day that its danger is fractional.

Compared to that of pre-LAVERAN-MANSON-ROSS days, it may be said that if a man gets malaria in modern times the fault is with the man, because he did not follow the precautions now so well known.

No insurance company, I take it, would have insured the lives of Drs. LOW and SAMBON when they went to live in the Roman Campagna during the most malarial periods of the year, yet they came back without being infected and in excellent health. The mosquito-net was the sole protector. So it will be in all malarial lands. Anyone can defy malaria who wards off the mosquito. And not only malaria, for the U.S. Army Commission has proved that yellow fever can be prevented in a similar manner. In the West Indies, therefore, where malaria and yellow fever are the scourges, there is no reason why one should contract either of these diseases. So keenly has this come home to us, that many declare that the tropics are dangerous to the white man only through their diseases, not through their climate. Ward off malaria, and health is capable of being enjoyed in any climate. Whilst not, perhaps, going so far as to say that climate has no deleterious effect, I do claim that in

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the tropics, if malaria can be prevented, the white man can resist the effect of a hot climate for an indefinite time.

In temperate climates we have to build our homes so as to keep out cold, damp, and thereby protect us from lung troubles, so the tropical resident has to build his house a "protected house" to keep out the mosquito.

Beyond malaria, which involves the insurance companies to but an infinitesimal extent nowadays, the other disease of importance is dysentery and its sequelæ. That is, again, greatly lessened to-day by the emetine treatment of ROGERS. We are quite aware that emetine leaves a tendency to post-dysenteric colitis, perhaps to a greater extent than the ipecacuanha treatment of pre-emetine times, but the death-rate from acute amœbic dysentery has been lessened to a marvellous extent, and although colitis is perhaps more prevalent as a sequela, the man is sent home as an invalid, and the tropical region where he contracted the disease is freed in its statistical records from the old-time death-rate from this disease.

What, then, is an extra premium charged for, as other diseases are but on a platform with home ailments? These facts I have brought forward on several occasions, and whether in consequence of my speaking and writing I know not, but several companies now advertise world-wide policies. In a short time all companies must follow suit, otherwise the world-wide policies will kill out the still perverse companies.

The great diseases of the tropics affect, for the most part, the young men—the unacclimatised, as they are called. It is during the first two or three years of tropical life for young men going out that the tropics is dangerous, and the younger they go out the greater the danger. Time was when men went out at a younger age than now. Sixteen or seventeen was a common age to send out young men some thirty years ago. Gradually the advantages of sending young men out at about twenty became apparent, and for a man to go out before twenty-one is looked upon to-day as inadvisable and dangerous. Twenty-three is the better age, and twenty-five the best. This fact, that the earlier the age the more the danger, affects insurance. Before going out the young man is likely to be insured by his parents or guardians; and as the younger the age the better the terms of the policy for home life, he may

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be insured, say, in the office at home when seventeen When, however, he goes overseas to the tropics, the extra-premium question arises. Abroad, his salary barely keeps him, and he has £10 or £20, or in some cases even £40, charged him, over and above the home premium. He is now free from parental help and control, and the extra charge falling upon him either runs him into debt or he lets the policy lapse, and the money he paid is lost to him and given to the benefit of the company, "for the risk they have run."

I have brought forward more than one plan whereby this dropping of policies may be prevented. Of these attempts to lessen the chance of the young man allowing the policy to lapse, I suggest the following scheme as one which would accomplish this:—

Supposing a young lad of seventeen is insured, whilst working in an insurance office at home, for £1,000. When he is twenty years of age he is sent overseas in the company's service. Up to then his father paid his insurance, but when he goes abroad an extra premium is imposed and his father expects him to pay the premium on his life. He finds after a year or two the premium, plus the extra premium imposed, is more than he can meet and he allows it to lapse.

To avoid this occurring, I would suggest that instead of the company charging the extra premium, that the company reduce their liability, and that, instead of being liable for the £1,000 originally taken out, the liability of the company be reduced to, say, £500 in case of the death of their client, or it might be to £750 if the climate is a "good" one. This should hold good for say five years, that is to say, until the insured has passed through the "dangerous period" of his tropical residence. If at the end of the five years, and if the client can produce a doctor's certificate to the effect that he is quite well I suggest that the extra premium be abolished altogether, or at least reduced by half, and their full liability of £1,000 be restored. Instead of these hard and fast lines being followed, the restoration might be graded. If it is reduced, to begin with, by £500, after a period the liability might be raised to £700, and, after a few more years be graded up to £1,000.

The authorities of the insurance office scout this proposal as they scout any proposal made by outsiders, and especially by medical men. The actuaries are the sole authorities, and their laws and opinions are those of the Medes and Persians.

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I have been told by members of insurance companies whom I invited in 1908 to an address I was giving, that it did not matter what I said, however good it might be, that I was fighting a "close" body; that by to-morrow noon, the messengers from the various offices would meet each other and agree whether the office were to take any notice of the scheme or not—of course, they were not.

This, however, did not stop me, and I have lived long enough to see several of the measures repudiated by them at the time, now in full play. The change was an actuarial one, and not as a consequence of interfering doctors and other men ignorant of the calculations of the mathematically evolved schemes necessary to regulate insurance. The doctors have the practical side of the consequence of insurance before them and do not need mathematics to help them to conclusions. The doctors are the sole ultimate authorities, and no juggling with figures can alter their opinions.

When I read my paper before the Life Insurance Medical Officers' Association in 1903, Sir DYCE DUCKWORTH was in the chair, and he, in commenting upon my paper, said, "Mr. CANTLIE'S plan of dealing with extra premiums for young men would free the young man from increasing his payment, and at the same time diminish the risk of the company satisfactorily. I do not know if business men would accept the principle, but to me it seems feasible and fair. I happened to see the manager of my own insurance office to-day, and I put this to him. He said it was nonsense on the face of it, and that no insurance office would contemplate such a matter as sound business. He went on further to repudiate several of the assertions of Mr. CANTLIE throughout this paper, which he had read, and to say that these matters do not, as Mr. CANTLIE says, rest entirely on the *dicta* of the doctors." He says, "I would recommend British insurance offices to rely for advice as regards the value of the lives of tropical residents on the medical officers of their companies, and not on their actuaries. The actuary cannot know, he has no means of knowing, the effect of climate or the local state of healthfulness in any individual colony." Then he goes on to say that "the actuary's figures are misleading, and his statistics of no use to him there. To-day I was told that that was not the case. I was told there was a great deal of mathematics in these cases, and that it is not merely an opinion formed on mere climate which the person goes to,

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but there are actuarial matters of great importance which we doctors are not supposed thoroughly to understand." Something wants doctoring, and it is not the doctor, but—

The Insurance of Natives.—The question of insuring the lives of natives remains a vexed one. British offices, for the most part, do not push this business; American insurance offices do. I am not prepared to give you a statistical table of the expectancy of life of, say, a Hindoo, Malay, Chinaman, or negro: no statistics are available; but I am in a position to express an opinion in regard to the matter. The question narrows itself down to a very fine point. It is only the more advanced and intelligent of the natives of any Asiatic country who take out insurances on their lives. It is the natives who have become "Anglicised" to a greater or less extent that usually come forward for insurances. But in the process of becoming Anglicised they have acquired more than an imperfect knowledge of English and British habits in business. They affect British ways of living to some extent, and especially do they take to the food and drink which they see supplied to the British table. Beef or mutton take the place of fish or fat pork in the diet, and European wines, especially champagne, are the constant concomitants of, say, the Chinaman who takes to European "chow." A rich Chinaman who affects the European mode of living has his Chinese "chow" with his family, and European "chow" (it may be immediately afterwards) with his Anglicised *confrères*. The excess of rice at one meal, with beef, etc., and sweet champagne—the sweeter the better he likes it—in time tells its tale. Gout, kidney and liver troubles supervene, and his days are shortened. It is from this very class that most native lives are insured, and they are the very worst lives of the land they belong to. The ignorant and simple living native might be as good a life as any European—as a matter of fact he seldom is—but when he departs from his simple habits, and takes to "foreign" ways of eating and drinking, his life is not a good one. I saw much of American methods of dealing with the Chinese in regard to insurance. At one time, in Hong Kong, the Chinese insured largely in American insurance offices, but during the last years of my stay there they almost ceased doing so. Perhaps they have taken it up again, but I hope on a different footing to that which was in vogue when I knew the methods followed.

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Insurance Anomalies.—The insurance offices say, "Well, we slump the risks, and what we lose by the younger men we gain on those of more mature years." Many hardships are, however, incurred therefrom. But yesterday I saw a nurse proceeding to one of our colonies, a healthy woman of forty, who had to pay an extra premium of £10 a year; her actual annual salary amounting to about £60. I know also of *patients*, who, on being recommended to go to a warm climate by their physician, had to pay an extra premium to their insurance offices—a most anomalous state of affairs. They were being sent to the only place in the world where health could be obtained and where their lives could be prolonged, yet they are asked to pay an extra premium for their doing so.

I have known men who, after long residence in a warm climate, find they cannot keep their health at home, and go abroad again to live. They have paid an extra premium on their life assurance whilst abroad; this has been rebated when they came home, but again enforced when, in order to live at all, they have gone to the part of the world which suits them best; the insurance office in this instance remitting the extra premium in the only place where their client could not live, namely, at home.

Discussion.

Sir William Leishman expressed the pleasure with which he had listened to the President's excellent address, which was full of common sense. It was up to the tropical members of the profession to put the insurance companies right. Actuaries took the line they did presumably because of defective information. Sir James said there were no proper statistics extant, but the speaker was doubtful as to that. He believed careful delving among the records of some of the Crown Colonies would produce such statistical evidence as might persuade the actuaries that the supposed danger to young life in the tropics was greatly exaggerated. It was very much to the detriment of the Empire that such an impression should be widespread. If the words of Sir James Cantlie should reach and impress the insurance companies concerned, they would have done much good.

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Sir LEONARD ROGERS said the PRESIDENT had raised one or two points of practical importance. One was as to whether the Society should form a Committee which could give information relative to insurance. A very important factor was the enormously improved prospects of healthy life in the tropics now as compared with not very many years ago, owing to the great advance in tropical medicine; for instance, inoculation against typhoid. In Calcutta he found that 80 per cent. of the attacks of typhoid in the General Hospital, among Europeans, were in the first three years of their residence there. That had been the greatest cause of death in India and other parts of the tropics, and was now easily preventable. The same could be said of cholera, dysentery and liver abscess; and if the insurance companies would take cognisance of these facts and reduce their rates, it would be a matter of great practical importance.

Another point was as to Americans being so far more enterprising than English companies. Some years ago he spent much time in analysing 4,000 post-mortem records in Calcutta, working out the causes of death and the implication of various organs, and he never once got a reference to that work from any English insurance company's officer; but he had heard from one of the biggest American companies that it had reprinted the whole report of the investigation for the use of their officers in America.

The following gentlemen also took part in the discussion :—Lieut.-Col. CLAYTON LANE, Lieut.-Col. J. C. KENNEDY, Squadron-Leader H. E. WHITTINGHAM, Drs. LEOPOLD HILL and A. T. OZZARD, and Messrs. H. E. HORNBY and A. L. SHEATHER, and the PRESIDENT replied.

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SHORT PAPERS.*

NOTE ON RELATION BETWEEN TSETSE-FLY *(GLOSSINA MORSITANS)* AND GAME IN THE PROCLAIMED AREA, NYASALAND.

BY WILLIAM A. MURRAY, M.B., CH.B.

The Glossinæ are fairly widely distributed over British Nyasaland, but are chiefly found in the narrow forest belt which covers the west shores of Lake Nyasa, from Chintechi in the north to Fort Johnson at the south end, then extends along both banks of the Upper Shiré as far as the Murchison Cataracts opposite Blantyre (see map).

Of these, *Glossina morsitans* is by far the most important and the most widely distributed species. *Brevipalpis, fusca* and *pallidipes* are also found, but more rarely.

During the writer's residence of twelve years at Mvera, Central Angoniland, on the border of this fly-infested forest, he had an opportunity of carefully observing the fly and its movements in the section of forest adjacent, extending roughly for fifty miles from north to south and twenty-five miles in width.

When the writer first arrived at Mvera Station (which is situated just outside the western border of the forest, and is about twenty-five miles west of Domira Bay) he found the whole adjacent section of forest (which was afterwards proclaimed as the Quarantine Area for Sleeping Sickness) quite clear of tsetse-fly, except at one spot near the south-east corner of this area. At the same time there was very little game to be found in this forest, except at the same place.

Enquiries from old residents at Mvera elicited the fact that prior to 1896, when the rinderpest swept through the land, and decimated alike native cattle and all kinds of large game, the latter had roamed freely over practically the whole of the forest, and that wherever game was found there was also tsetse-fly. This information was confirmed by

*With a view to making the TRANSACTIONS more interesting, it is proposed to insert SHORT COMMUNICATIONS from Fellows relative to any matters of medical, sanitary or parasitological interest. It is felt that many Fellows in various parts of the world must be in a position to contribute short papers on these subjects, and the Secretaries will be pleased to receive any such from them.

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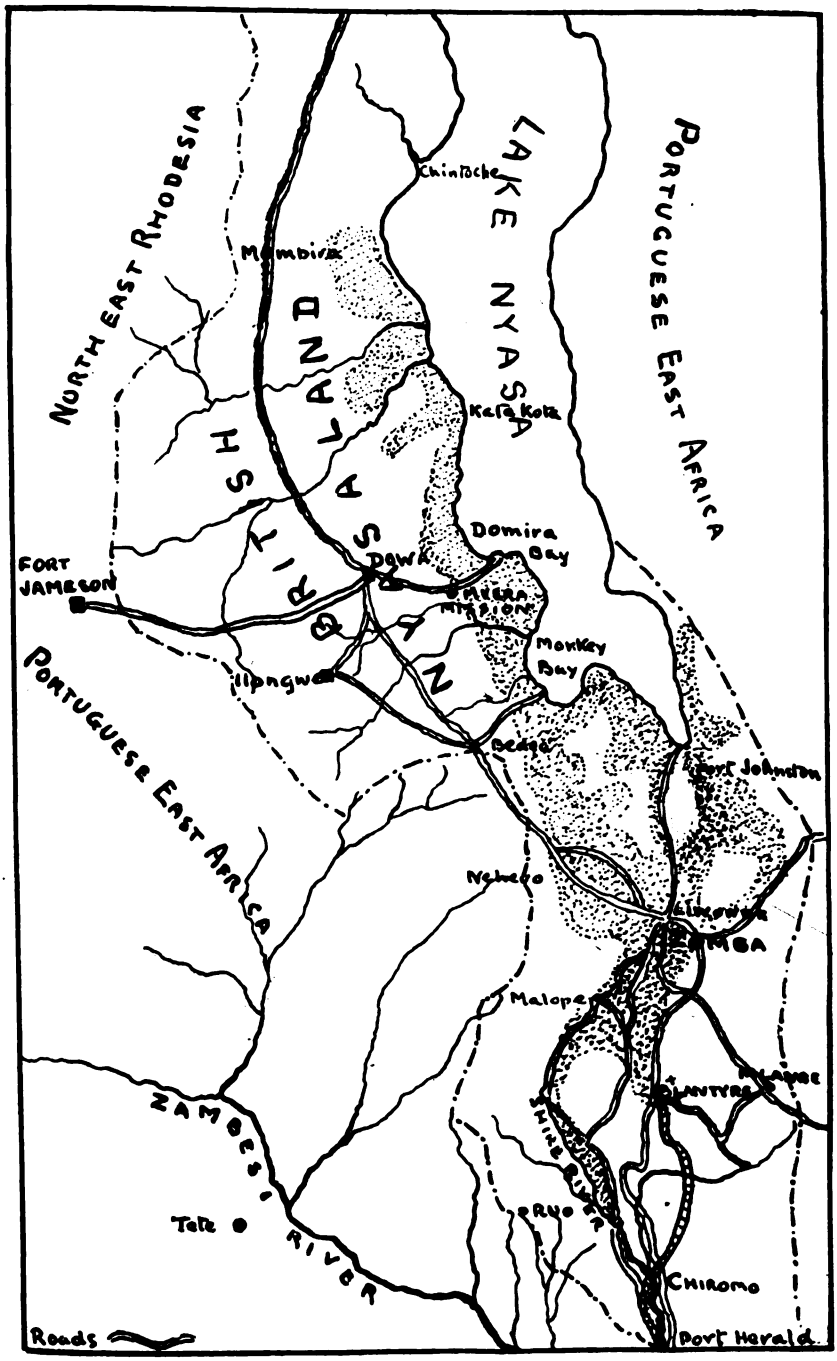

Map shewing TSETSE FLY AREAS in NYASALAND

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Sir ALFRED SHARPE, then Governor of the Nyasaland Protectorate, during a conversation with the writer at Dowa, in 1902.

Sir ALFRED had been a mighty hunter in the early years of the Protectorate, and well remembered that part of the forest for the abundance of both large game and tsetse-fly.

It was clear, therefore, that the rinderpest had not only almost wiped out the game, but that the tsetse-fly had also diminished to a corresponding extent all over that large area. It would be of great interest if one could find out whether the fly had died as a result of imbibing the blood of sick and dead animals, or whether they died off, or departed elsewhere, because of the subsequent great scarcity of mammalian blood as food. It may be said that, prior to 1900, the whole forest was very sparsely inhabited by the Achewa natives of the western plateau, owing to their dread of their hereditary enemies the slave-hunting Yaos, who live all along the lake shore.

The small herds of buffalo, eland, kudu, hartebeest and sable that had survived the rinderpest mostly took refuge in the Tsanjoka Marsh, some ten miles south of Domira Bay, and for some years until about 1904, practically confined themselves to that locality. Buffalo, eland and sable were also found at the Lingadzi River, near the northern boundary of this area, but there were no tsetse whatever at that time at the Lingadzi. As, however, game increased in numbers, and as also natives began to establish villages near both these localities, the game commenced to scatter over a wider area of forest, and gradually returned to their old haunts over the greater part of the forest.

The fly which had been strictly limited for six or eight years to the Tsanjoka Marsh, spread concurrently with the game. This was of great concern to the Europeans at Mvera, whose cattle were accustomed to graze down in the forest, and who depended mostly on donkeys for transport while travelling about in the forest. They found from 1904 onwards that the tsetse-fly free area became gradually more and more restricted, until by 1908 the fly was seen within a mile of Mvera Station. This experience was confirmed by the loss of several donkeys and cattle from nagana disease (the type was *T. pecorum* in all cases examined by the writer).

The foregoing account of the diminution and spread of tsetse-fly over a comparatively small section of the lake shore forest serves as an

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example of the close connection between the movements of game and tsetse-fly over the whole Nyasaland fly area. This, of course, only applies to the low-lying parts of that forest, as beyond an altitude of 3,000 feet tsetse-fly was rarely found in Nyasaland, even though game was abundant in otherwise suitable country.

The writer's experience, therefore, fully corroborates the paper by Mr. W. KIRBY GREEN, published in the *Minutes of Evidence* taken by the Parliamentary Commission on Sleeping Sickness, in June, 1914, p. 253.

HISTORY OF THE INTRODUCTION AND SPREAD OF HUMAN TRYPANOSOMIASIS (SLEEPING SICKNESS) IN BRITISH NYASALAND IN 1908 AND FOLLOWING YEARS.

BY

WILLIAM A. MURRAY, M.B., CH.B.

Late Captain S.A.M.C. and Officer-in-Charge Sleeping Sickness Investigation in Zululand.

INTRODUCTION.

The writer arrived in Nyasaland in March, 1901, and was in charge of a Native Mission Hospital at Mvera, about twenty-five miles west of Domira Bay, on the west shore of Lake Nyasa, until December, 1912, and had unusual facilities for observing the incidence and spread of human sleeping sickness in Nyasaland. Not only were all the first cases associated with his district, but he was asked by the Nyasaland Government to take charge of the Sleeping Sickness Isolation Camp for eight months during 1910 and 1911, and in 1912 was permitted, through the kindness of Colonel (now Surgeon-General) Sir DAVID BRUCE, C.B., to associate himself with the investigations carried out by the Sleeping Sickness Commission at Kaso, near Mvera. The results of their labours were published in 1914 as the *Sixteenth Report of the Sleeping Sickness Commission of the Royal Society*, and to this valuable report I am indebted for many of the following facts and references. For other data I am indebted to the official *Nyasaland Sleeping Sickness Diaries*

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(Nos. 1 to XXV., published between November, 1907, and December, 1915, and then discontinued).

WAS SLEEPING SICKNESS ENDEMIC IN NYASALAND OR INTRODUCED?

The Royal Society's Commission came to the conclusion that "Nyasaland Sleeping Sickness has not been introduced in to Nyasaland from Lake Tanganyika, but is an endemic disease of the country," basing their opinion on "papers by Drs. SANDERSON, SHIRCORE, and others." From this opinion I dissented strongly at the time, as Colonel BRUCE admits in his Report quoted above (*vide* pp. 34 to 38 for the argument *in toto*), and I have seen no reason since then to alter my conviction.

The following account of the manner in which the disease broke out in Nyasaland is part of the proof of my opinion that there was no sleeping sickness in Nyasaland prior to 1907 or 1908, and that it was introduced about that time from Lake Tanganyika or North-Eastern Rhodesia, or both.

HISTORICAL SURVEY OF SLEEPING SICKNESS.

Prior to 1884, human sleeping sickness (due to a protozoal blood parasite, *Trypanosoma gambiense*, and spread by an intermediary host, *Glossina palpalis*) was confined to the West Coast of Africa and the region of the Lower Congo. After the war against the Arab power in the Congo, in 1892, the disease spread widely over the Congo basin, due to large military movements. In 1896 the disease appeared in Uganda following the arrival of EMIN PASHA'S followers from the Congo Free State, who settled in Uganda. The disease spread rapidly about the shores of Lake Victoria Nyanza, and claimed about half a million victims between 1896 and 1904. By 1900 the disease had extended to Kasongo, south-west of Lake Victoria Nyanza, then spread to Lake Tanganyika and the Lualaba River towards the Katanga district.

At the end of 1907 the same disease (still caused by *T. gambiense* and carried by *G. palpalis*) had spread as far south as the southern shores of Tanganyika and Lake Mweru. By July, 1908, cases had been found along the Luapula River in North-East Rhodesia and near Lake Bangweolo. Investigation during 1907 and 1908 in North Nyasa districts proved that men of the Atonga tribes had been travelling in

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hundreds to and from Katanga and other infected districts of Tanganyika in search of work, so that there was every likelihood of infection being carried back by them to their homes in Nyasaland.

In 1908 cases of a more virulent type of sleeping sickness (later known as the Rhodesian type) were found in North-Eastern Rhodesia, near the Nyasaland border, and investigation showed that infection had taken place in or near the Luangwa Valley where no *G. palpalis* existed, but only *G. morsitans*, which up to that time had not been seriously considered a potential carrier of sleeping sickness.

The Nyasaland Government had meanwhile not been idle, but had closed the Rhodesian-Nyasaland Border in 1907 to all native travellers except at a few posts, where medical observation of all travelling natives was to be carried out, in order to prevent if possible the entry of infected natives into Nyasaland This measure proved futile, as the natives easily evaded the posts and the police.

Further, the Nyasaland Government Officers had, during 1907 and 1908, carried out careful investigations to find :—

(*a*) Whether *G. palpalis* existed within the borders of Nyasaland? None was found, but *G. morsitans* in abundance, also *G. pallidipes*, *G. brevipalpis*, and *G. fusca*.

(*b*) Whether any cases of sleeping sickness were to be found in any tsetse-fly area within the Protectorate or in neighbouring districts? Over 60,000 natives were examined for enlargement of glands, and the blood of all sick natives or those with enlarged glands was examined microscopically for trypanosomes, but not a single case of sleeping sickness was found before October, 1908.

For many years previous to this, the Government and Mission doctors scattered throughout Nyasaland (about thirty in all) had examined many thousands of sick natives annually, both clinically and with the microscope, and not a single case of sleeping sickness had been encountered. Had the disease been endemic in the country, I consider it impossible that one with such outstanding clinical and microscopical features could have been missed by all the medical observers in Nyasaland.

Personally, the writer had for nine years previous to 1910 examined not only many hundreds of sick natives annually from the very forest where the disease subsequently claimed so many victims, but had

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observed the health of thousands of native carriers, porters and other labourers regularly employed at Mvera Station, who were constantly traversing the portion of the neighbouring forest afterwards so heavily infected, without once meeting a case showing the marked clinical and blood features which became so common after 1910.

From 1905 he was on the look-out for cases of sleeping sickness, having been warned by accounts in the medical press of its ravages in Uganda and elsewhere.

Case 1.—In October, 1908, the first case of sleeping sickness in Nyasaland was discovered at Nkata Bay at the north end of Lake Nyasa. This patient, a native, gave a history of having travelled the previous year from his home in North Nyasa *via* Tanganyika to the Congo, and then returning *via* Cape Town to Nyasaland. His type of infection was never definitely determined, but was probably *T. gambiense* He continued for at least three years after in excellent health.

The Government resolved to establish an isolation camp for all cases that might be found, in a healthy fly-free area, and selected Dowa-Boma, about sixteen miles west of Mvera. Case 1 was accordingly brought by steamer to Domira Bay, and then conveyed in a hammock (unscreened) through twenty-five miles of forest swarming with tsetse-fly (*G. morsitans*) to Mvera, and thence to Dowa. He was accompanied by Dr. H., who was in charge of the new isolation camp, and by an armed guard. I subsequently questioned these Askari guards, who assured me that they were freely bitten by fly during the seven or eight hours they were passing through the forest. It may, therefore, safely be assumed that the patient was also bitten, and that there and then the first infection of the fly along that road occurred.

Case 2.—The second case resulted from the unfortunate accidental self-infection of Dr. H. from his patient at Dowa. As the writer visited Dr. H. and his patient at that time (November, 1908), he has no doubt whatever on this point. Dr. H. suffered from fever for some weeks, but considered it to be malarial, then improved, and undertook a visit to the forest along the Mvera-Maganga road to search for other species of tsetse-flies than *morsitans*. After about a week's sojourn in the forest he found himself too ill to work and returned to Dowa. In passing Mvera he complained to the writer that he had been badly bitten by tsetse-fly (*G. morsitans*) all the time he was in the forest. On his return to Dowa,

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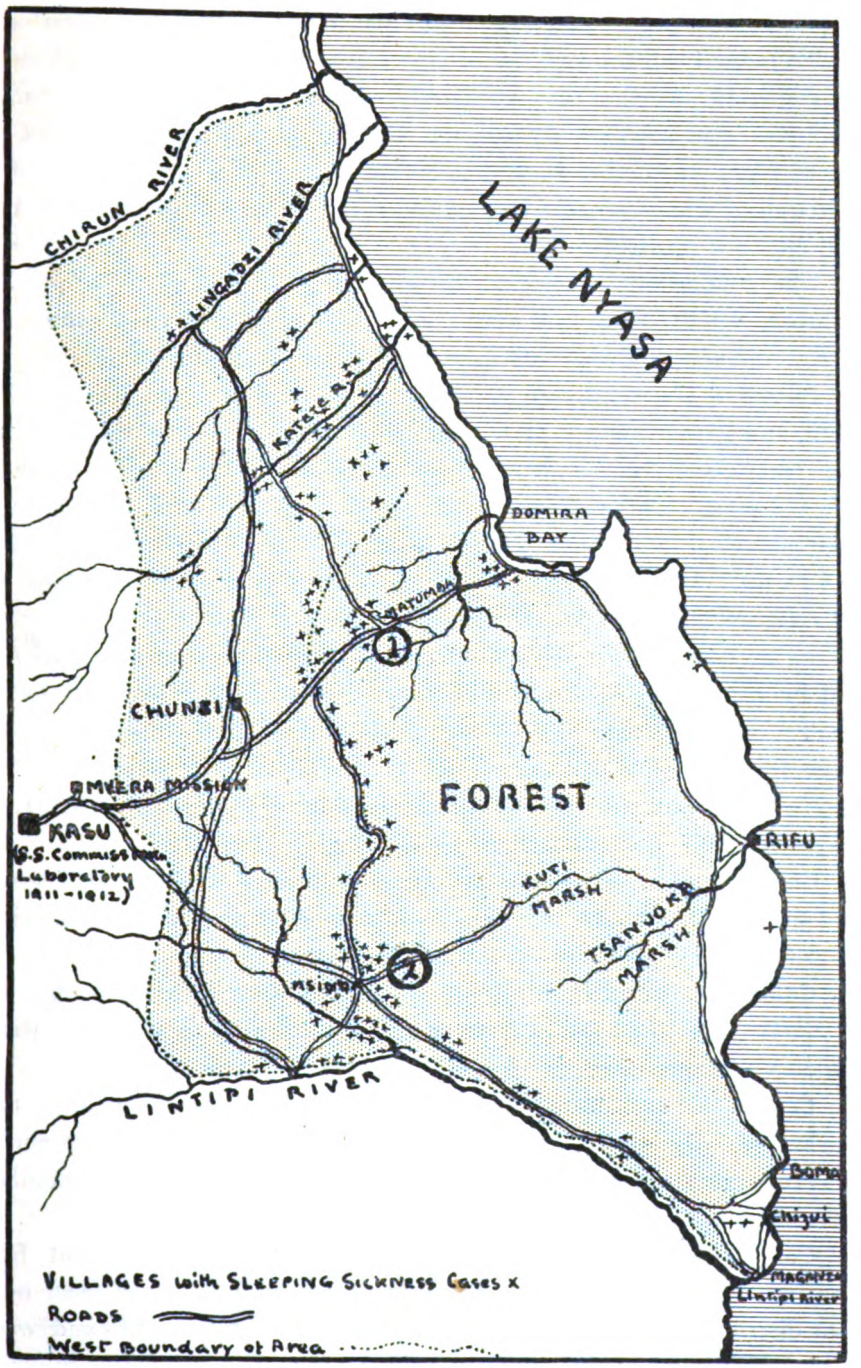

MAP OF SLEEPING SICKNESS PROCLAIMED AREA.

Shaded portion = Forest.
(1) Where first two cases are supposed to have infected fly.
(2) Where Case 2 (Dr. H.) was ill and infected more fly.

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trypanosomes were diagnosed in his blood. He died soon after.

To judge from the rapidity of his illness his infection resembled the *rhodesiense* type rather than the *gambiense*, but the case of Capt. TULLOCK who was also accidentally infected with *T. gambiense* in Uganda, and whose illness also ran a very virulent course, seems to show that a direct infection of the *gambiense* trypanosome into a susceptible human being will enhance its virulence.

The conclusions arrived at by Dr. DUKE in Uganda (*Parasitology*, October, 1919), as to the variation in virulence of trypanosomes after direct, and after indirect infection, would seem to confirm this.

Case 3.—The third case, a native, was found at Kota Kota in May, 1909, and gave a history of having some time previously returned from the Luapula River in North-East Rhodesia, a known *T. gambiense* infested area. This patient was also taken to Domira Bay by water, and then conveyed in a similar manner to Case 1 through the forest to Mvera and Dowa. It may again safely be assumed that more tsetse-flies were infected on this occasion along that road, and that the infection was spread by means of game, which is fairly plentiful in that neighbourhood, and by natives living in that vicinity. It is noteworthy that the first locally infected cases, and most of the subsequently discovered cases, came from these two original foci, viz., villages near the Domira Bay-Mvera roads, and along the Mvera-Maganga road, where Dr. H. had stayed for some time during his illness.

Cases 4 and 5.—Cases 4 and 5 (native) were discovered at Blantyre, and the locality of their infection could not be determined, as information as to their previous movements was too vague.

Cases 6, 7, 8 and 10.—These four natives were infected during 1909 in North-East Rhodesia, at or near Luangwa Valley, the greater part of which was soon after found heavily infected by *T. rhodesiense*.

Case 9.—Was infected about December, 1909, while walking along the Nyasa Lake shore, through the forest between Domira Bay and the Maganga road. He was only discovered six months later at Zomba to have sleeping sickness.

Case 11.—Was the first case found locally infected in that forest. He lived near the Mvera-Domira Bay road, and had never been out of that district. He was brought to my hospital for treatment, and was at once recognised as suffering from a disease differing from any previously

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seen in the district, but resembling the sleeping sickness cases then at Dowa.

Case 12.—Was a European, who had arrived a few weeks before from Cape Colony, and who received his infection in August, 1910, while hunting for a few days in the vicinity where Dr. H. had been ill nearly two years before.

The Government now detailed two medical officers to examine the natives of this portion of the forest thoroughly, and some thirty further cases were discovered in villages within a 20-mile radius of the centre of the Domira Bay road, most of them living near the two centres first infected. The infected area of forest bounded on the north by the Chirua River, west by the edge of the forest, south by the Lintipi River, and east by the lake shore, was proclaimed a quarantine area, and all movements of natives to and from this area were stopped.

The inhabitants of contiguous fly districts to the north and south of this area were carefully examined for cases of sleeping sickness, but not one was found until several months later a few cases of recent infection were found near Monkey Bay, about twenty miles south of the quarantine area. Other tsetse areas beyond those in the North and South Nyasa districts were, after careful medical examination of their inhabitants, declared still free of the disease in June, 1912. Soon after this date, one case occurred in the Kota Kota, and three cases in the Upper Shiré districts. Gradually the disease was found to spread more and more widely, until by 1915 practically the whole of the *morsitans* infected area of Nyasaland (stretching in a narrow belt along the west shore of the lake from Chintechi in the north, to the Murchison Cataracts on the Shiré River in the south) was found to contain infected fly, game and natives, but the degree of infection nowhere seemed to become as intense as in the part first infected near Domira Bay. Between October, 1908, and December, 1915, about 250 cases of sleeping sickness were discovered in Nyasaland. Of these only three survived for one or two years after infection, and only one lived for three years or over.

During the war all sleeping sickness investigation work in Nyasaland was stopped, and little information is available as to the subsequent progress of the disease. As far as can be gathered, however, it would seem that the pathogenicity and spread of the epidemic have both diminished, and that few cases of infection are now to be found.

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A similar course of events has taken place in the Luangwa Valley in North-East Rhodesia, which was also found heavily infected in and after 1909, but where the disease has now died down to very small numbers annually.

It would seem, therefore, as if this strain (*T. rhodesiense*) in Nyasaland and Rhodesia was most virulent and spread most rapidly after its first introduction (about 1908 to 1910) into these countries, but that after many passages through the three hosts concerned (fly, game, and man), the infectivity or virulence or both has been much reduced. No opinion, however, can be hazarded as to the future until a thorough medical survey of the exact present prevalence of the disease in Nyasaland has been made.

It is not easy to explain the mode of introduction of *T. rhodesiense* into Nyasaland. According to the writer's hypothesis it would seem necessary to assume that the original source was *T. gambiense* from the north, and that by some transition at present not understood, and which has not yet been seriously investigated, the change of vector from *palpalis* to *morsitans* fly has resulted (probably after several passages through fly and game) in a different strain of trypanosome, which causes a more virulent type of disease, and can by other methods also be distinguished from the original strain.

In conclusion, the writer wishes to state that he thoroughly concurs in the conclusions arrived at by Sir DAVID BRUCE and his collaborators in Nyasaland as to the highly important rôle played by the larger varieties of game in the dissemination of the disease in Nyasaland, and in acting as a permanent reservoir of all the trypanosomes pathogenic to man and domestic animals.

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A NOTE ON THE TREATMENT OF SUPPRESSION IN BLACKWATER FEVER.

BY

A. F. WALLACE, M.D., *Medical Officer, Northern Rhodesia Administration.*

A European, W. J. C., was admitted to the Broken Hill Hospital, Northern Rhodesia, on May 29th, 1921, suffering from blackwater fever. He had had blackwater fever twice before—once on the Gold Coast in 1912, and again in German East Africa in 1913. During the war he had been an officer in the Flying Corps, and he returned to Rhodesia in 1920.

Since his return he had had one severe attack of malaria in 1920, and for the six weeks prior to his admission to hospital he had repeated attacks of malaria. As was to be expected, he was not a man who took his quinine regularly.

Blackwater fever developed on the evening of May 28th. I saw him next forenoon. He was removed to hospital the same afternoon by motor car, a distance of four miles. In hospital he was given the usual routine treatment: ten grains of sodium bicarbonate every two hours and copious drinks of plain boiled water. His temperature on admission was 103°. It was subnormal next morning, and during his stay in hospital it never again rose above normal. A blood slide taken the day that he was admitted showed no malarial parasites.

The following table shows the amount of urine passed daily :—

May 29th	(10 hours)	4	ozs.	June 5th	(ten hours	5	ozs.
„ 30th	„	10½	„	„ 6th	„	15	„
„ 31st	„	1	„	„ 7th	„	24	„
June 1st	„	0	„	„ 8th	„	61	„
„ 2nd	„	⅞	„	„ 9th	„	89	„
„ 3rd	„	2	„	„ 10th	„	146	„
„ 4th	„	3¼	„	„ 11th	„	141	„

It will be seen that during the three days from May 31st to June 2nd

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the patient had only passed $1\frac{7}{8}$ ozs. of urine. He had constant bilious vomiting, and was unable to retain anything by the mouth. Soon after admission, rectal salines were started, but after two days the patient gave up trying to retain them. On May 30th intravenous salines were commenced, and up to June 2nd over seven pints of saline had been injected. On June 3rd his condition seemed hopeless. Nothing could be given by the mouth or rectum, and there was marked œdema of the face, chest and arms. Hot-air baths, combined with injections of pilocarpine had been tried, and he had also been given injections of pituitrin.

At this critical period I found the following remark on the treatment of anuria in OSLER's *Practice of Medicine:* "Large hot irrigations with normal salt solution with Kemp's double-current rectal tubes should be tried, as they seem to stimulate the activity of the kidneys in a remarkable way."

Acting on this, two thin œsophageal tubes (size 20) were stitched together, side by side, and with the tip of one three inches lower than the tip of the other. This was then passed per rectum high up in the bowel. At first every hour, and later every four hours, four pints of as hot saline as the patient could bear were run in from a douche can. Shortly after each irrigation the patient had a desire to micturate, and it seemed as if the irrigations had a direct effect on the blocked kidneys. The urine was at first passed in teaspoonful amounts, but it rapidly increased until, on June 10th, he passed 141 ozs. The change in his appearance from a bloated puffy individual to a thin and washed-out one was very striking.

This rectal irrigation treatment does not appear to be mentioned in books on tropical medicine. It certainly appears a rational means of applying heat to the kidneys, and must be more effectual than the application of poultices and stupes to the loins.

It would have been more dramatic if I had commenced the irrigations on June 1st, when the patient was passing no urine at all, but I am convinced that this method should be given a trial in any case of suppression.

I have to thank Dr. AYLMER MAY, Principal Medical Officer, Northern Rhodesia, for permission to publish this note.

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A PRELIMINARY NOTE ON THE DEVELOPMENT OF *LOA LOA* (GUYOT) IN *CHRYSOPS SILACEA* (AUSTEN).

BY

A. CONNAL, M.D., D.P.H., D.T.M. & HY. (*Director, Medical Research Institute, Lagos*),

AND

S. L. M. CONNAL, M.B.E., M.A., B.SC. (*From the Medical Research Institute, Lagos*).

We spent two months of the present year (1921), from 28th May until 20th July, in Sapele, investigating the possibility of Chrysops being the carrier of *Loa loa*.

Sir PATRICK MANSON'S suggestion, made in 1895, and Professor LEIPER'S work, done in 1913, as well as our own preliminary efforts in 1917, all pointed to the likelihood of this insect being the intermediate host of the filaria.

Sapele is a trading centre on the river Benin, two days by launch east of Lagos, through the creeks. Our operations were confined to the opposite side of the river from Sapele town, were Messrs. Miller Brothers, Ltd. (of Liverpool), have a large rubber estate. Bounded by swamps as it is, and with a large labouring population, it formed an ideal site for our purpose, particularly as Mr. J. B. Methven, the manager, took a warm interest in our work, besides providing us with a bungalow and freely offering every facility. Chrysops were extremely numerous, and the percentage of labourers harbouring *Loa loa* was high. We conducted twenty feeding experiments, in which we fed six hundred and eight flies. *Chrysops silacea* predominated, and the remainder were *C. dimidiata*. Five native boys were selected as a source of the infecting feed. Three of these were chosen because the number of embryos in their blood was high, and the other two because the number was small. In all five cases, as far as one can judge by repeated (hourly) examinations of the blood on different days, the infection was a pure one.

Most of the flies used in the experiments have been preserved for section. These, except in a few instances, have not yet been examined.

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One hundred and fifty-three have been dissected and each one was found to be successfully infected. The findings have been so consistent and so similar in the different series of experiments, that we feel justified in making the following preliminary statements, particularly as some considerable time must elapse before we can have all our preserved flies sectioned and examined.

Within six hours of the infecting feed, the great majority of the embryos have cast their sheath and migrated through the stomach wall to reach the abdominal muscles, where most of them come to rest. A number proceed to the muscles of the thorax and of the head. Within twenty-four hours, in a heavy infection, the young filariæ may be found in all these situations.

The embryos do not develop with equal rapidity. In some of the experiments, examinations were made at six-hourly intervals up to seventy-two hours after the infecting feed, and, in diminishing numbers as the time advanced, up to forty-eight hours, embryos which had not undergone ecdysis were found alive and active. A few die in the stomach without having cast their sheath, and a small number succumb after ecdysis. The first change, after the sheath is shed, is a shortening and broadening. Growth is rapid, but up to the third or fourth day the shape is cigar- or torpedo-like. By the fifth and sixth days, the young filariæ may be seen, considerably elongated, lying coiled up in the muscles, giving a striking appearance of whorls. All the forms are not of equal length on any given day. This difference in size is attributable, for the most part, to the difference in the time when the earliest forms migrated through the stomach wall, some coming to rest in the muscles at least forty-eight hours before others.

The preference for the abdominal muscles and the grouping in whorls are very outstanding features.

We have not observed any stages where motion entirely ceases, as is described for *Filaria bancrofti.*

The final stage of development is reached in from ten to twelve days. At this time the individuals in the abdomen and the thorax begin to travel towards the head. Some may be found in the roots of the wings and in the coxæ. They have been found even as far down as the metatarsus. It seems likely that this was due to overcrowding. When ready for another host, the fully metamorphosed forms are accumulated

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at the root of the proboscis and in the labium. They gain exit by the labella.

In attempts to infect various animals (five guinea-pigs, two rabbits and a monkey), it was noted that, in a heavy infection, the fly, whilst feeding, shows signs of irritation, withdrawing and reinserting the proboscis several times. Finally, it jerks out the organ and rapidly "milks" the proboscis with the fore-legs, flicking the minute masses of filariæ some little distance away, and distributing others by moving its position. In slight infections it is difficult to detect the issue of the worms, a fleeting impression of a glistening movement being sometimes obtained. When the filariæ have reached the skin, they are just visible to the naked eye. Their movements are extremely rapid and in less than sixty seconds they have completely disappeared beneath the epidermis.

We are not able to state how long a fly may remain infective, as none of our specimens survived the infecting feed for a longer period than fourteen days. It is interesting to note, however, that of three flies found during their life-time to be doubly infected, none lived longer than one day after filariæ were observed issuing from their proboscis.

Further description is reserved until all our material has been studied.

The flies used in the experiments were caught wild in the bush. Only 30 to 40 per cent. of the individuals in one batch would feed on the day of capture. Only those flies whose stomachs were empty were allowed to feed. This precaution excluded the possibility of a natural infection contracted less than two days before, as even a small feed of blood can be readily detected through the abdominal wall, and it takes at least forty-eight hours to disappear. The remaining 60 to 70 per cent. were set aside, and some were used a day or two later for another feeding experiment. Most of them, however, were dissected on the same day or were preserved for future dissection. Still others were fed on a clean guinea-pig or on a non-infected native boy, and dissected at definite intervals. By these means we established some manner of control. The total number of controls examined so far is two hundred and twenty-five, and the number found naturally infected is eight, which is just over 3·5 per cent. In the one hundred and fifty-three experimentally-infected flies examined, five showed a double infection, that is, just over three per cent. had a naturally acquired infection before being again infected by us.

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As regards the bionomics of Chrysops, we were successful in finding the eggs and the larvæ but not the pupæ. The eggs were found laid in a batch on a moist dead leaf lying on the surface of the mud of the swamp. Larvæ in different stages of development could always be found, but never close together, in the mud of the swamp, at a depth of a few inches. Very young larvæ were also found in the moisture between the leaves of the "screw-pine" and in the flower of a shrub growing on the river banks. As none of our specimens has yet pupated, we are not able to state which species prefers the mud of the swamp, or if there is any preference. We were able to induce only one captive fly to lay eggs.

C. silacea in Sapele far outnumbers the other species. *C. dimidiata* is fairly common. In nature it is very distinct from *C. silacea* in size, in colour, and in markings, and is differentiated even by the native *C. longicornis*, in our experience, is rare.

No other species of Chrysops were obtained. As regards the habits of the flies they are most troublesome in the late morning and in the early evening. They are practically noiseless, very persistent, and it is usually the act of withdrawing, not of inserting, the proboscis that is felt. They are voracious blood-suckers and seldom leave off until enormously distended.

Note by Editor.—KLEINE'S reference to the development of *Filaria loa* in Chrysops appears to have been overlooked by these observers. The exact reference and review in the *Tropical Diseases Bulletin*, 1916, vol. 7, p. 361, is as follows :—

KLEINE, (F. K.). **Die Uebertragung von Filarien durch Chrysops** (The Transmission of Filaria by Chrysops). Zeitchr. f. Hyg. u. Infektionskr. 1915, Oct. 26. Vol. 80, No. 3, pp. 345-349.

This is an account of an investigation, carried out at Eseka in the Cameroons, regarding the transmission of Filaria by Chrysops. The inhabitants of the district in and around that in which the work was carried out are heavily infected with *Filaria loa*, and Chrysops and sand-flies abound.

Six hundred female flies were examined, and, of these, five hundred were *C. dimidiata* and the remainder *C. silacea*. Of the whole number, thirty-two were found to be infected, *i.e.*, 5 per cent., and in the case of nine of these infected flies the larvæ were fully mature.

Development, according to the author's account, appears to take place in the fatty connective tissue surrounding the tracheæ in the abdominal cavity, and it is to that site that the embryos converge when they first perforate the gut of their host. In the early stages the larvæ remain motionless, and it is not until their evolution has made marked advance that they commence to leave their position in the fatty connective tissue and to wander about the abdominal cavity. Later, they make their way to the cephalic end of the body, and, in two cases, the author was able to demonstrate the emergence of the larvæ from the proboscis. This was carried out by placing the head, after removal, in a few drops of salt solution and observing under a low power of the microscope.

The size of the mature larva is given as being the length of the diameter of the field, when ocular 2 and objective AA are used, with the tube drawn out to 160mm.

[The Chrysops used in this work were, apparently, collected in a haphazard fashion, and the hosts upon which they had fed were consequently unknown. The nature of the larvæ, therefore, would appear to be undetermined. Neither illustrations nor a description of the larvæ are given, and no attempt was made to communicate filarial infection by means of infected flies. The work must be regarded as incomplete.] R. P. C.

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EXTRACTS FROM THE TROPICAL DISEASES BULLETIN.

PEREZ GROVAS (P.). **Experimental Transmission of Yellow Fever Cases occurring in Vera Cruz in 1920, together with the Isolation of** *Leptospira icteroides*.—*Jl. Amer. Med. Assoc.* 1921. Feb. 5. Vol. 76. No. 6. pp. 362-365.

The chief importance of this paper lies in the fact that it gives the first confirmation of NOGUCHI'S work on *Leptospira icteroides* of yellow fever. On the occasion of an outbreak of the disease in Vera Cruz in July, 1920, after a complete immunity for many years, the writer took the opportunity of repeating some of NOGUCHI'S experiments. The result has been that guinea-pigs have been successfully inoculated with the virus of yellow fever, while the characteristic lesions in these animals and the leptospira itself, as described by NOGUCHI, have been found. The strain was easily passed from one guinea-pig to another, and cultures of the organism were also obtained. These cultures were proved to be virulent to guinea-pigs.

The paper is in the nature of a preliminary report, and a more detailed account of the investigations, which are being extended, is promised. C. M. W.

i. LANFRANCHI (A.). **Sul possible passaggio dei tripanosomi nel latte.** [On the Transmissibility of Trypanosomes in the Milk.] *Atti. R. Accad. Lincei. Rendiconti.* 1916. (5 ser.) Vol. 25. (1 sem.) p. 369.

ii. LANFRANCHI (A.). *Idem.* [2nd note.] *Ibid.* 1918. Vol. 27. (1 sem.) p. 62.

In these two communications the author records the results of some interesting experiments undertaken to ascertain whether trypanosomes can be transmitted from an infected mother to her offspring through the milk.

[The first has already been briefly noticed in this *Bulletin*, Vol 8, p. 275.]

i. The first experiments were made with dogs, using four different species of trypanosome. The method employed was as follows: A bitch was inoculated, immediately after parturition, with 2 c.c. of the citrated blood of an infected guinea-pig. Her milk was afterwards tested, at intervals, both by direct microscopic examination and by intraperitoneal inoculation into rats and mice, in order to ascertain whether it contained trypanosomes. Her pups were also examined, by the same methods, to discover whether they had contracted her infection. The strains of trypanosomes used were obtained from the Institut Pasteur.

1. *T. brucei.* In the milk of the bitch inoculated with this organism no trypanosomes were ever discoverable microscopically, but one of the rats into which it was injected became infected, and died of trypanosomiasis. All the four pups of this bitch died, and were found to have acquired their mother's infection; for although the blood of all gave negative results when examined under the microscope, some of the rats inoculated from each pup became infected with *T. brucei.*

2. *T. rhodesiense.* All the pups belonging to the bitch infected with this trypanosome remained healthy. No trypanosomes were demonstrable in them microscopically, and none of the mice into which their blood was injected acquired infection. The milk of the mother was always negative microscopically; but on one occasion a mouse into which it was injected gave a positive result—acquiring a fatal infection with *T. rhodesiense.*

3. *T. evansi.* The milk of the bitch inoculated with this trypanosome remained completely negative to all tests. Her pups also all gave wholly negative results—in trypanosomes being discoverable in their blood microscopically, or by animal inoculation.

4. *T. gambiense.* Of the seven pups belonging to the bitch inoculated with this trypanosome, six gave no evidence of infection; but one acquired its mother's infection, for injection of its blood into a mouse gave a positive result. The milk of the mother was

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microscopically negative throughout, but on one occasion its injection into a mouse was followed by a positive result—the mouse succumbing to a *T. gambiense* infection.

From the foregoing experiments the author concludes that *T. brucei*, *T. rhodesiense*, and *T. gambiense* can pass into the milk of an infected bitch; and further that, in the case of *T. brucei* and *T. gambiense*, the mother can transmit the trypanosomes to her offspring in the process of suckling them.

ii. The second paper describes further experiments made by the same methods but using different animals for inoculation. The same four species of trypanosome were employed, and in addition the strain which the author calls "*T. lanfranchii*"—the organism with which he himself accidentally became infected. [See this *Bulletin*, Vol. 8, p. 274.]

1. *T. brucei*. Three female rats and one guinea-pig almost entirely negative results after inoculation with this trypanosome. All their offspring remained uninfected. The milk of the mothers gave negative results on all occasions save one, when one of the mice injected with the milk of one rat proved positive.

2. *T. rhodesiense*. Two rats and a guinea-pig were inoculated (the latter before parturition) with this trypanosome. Microscopic examination and inoculation tests carried out with the milk of the mothers and the blood of the offspring all gave completely negative results.

3. *T. eransi*. Two female rats and a guinea-pig inoculated with this species again gave results which were chiefly negative. All the offspring remained uninfected. The milk of all the mothers appeared negative to microscopic examination; but its injection into other animals gave positive results on two occasions—samples of the milk of one rat and of the guinea-pig each once causing infection of mice into which they were injected.

4. *T. gambiense*. Similar experiments carried out with this trypanosome, injected into two rats and a guinea-pig, gave negative results throughout. No evidence was obtained indicating that the trypanosomes had passed into the milk or the young.

5. "*T. lanfranchii*." Two strains, isolated at different times from the author's blood, were employed. Two rats and two guinea pigs were inoculated. All tests of the milk and the offspring were negative with two exceptions. On one occasion the milk of one of the mother rats gave a positive result when injected into mice; and one of the young guinea-pigs died, its blood, when injected into rats, proving positive. All the other young apparently remained uninfected.

A cat, which gave birth to a single kitten, was also inoculated with this strain. No trypanosomes could be detected afterwards in the cat's milk or in the kitten, either by microscopic examination or by animal inoculation.

The same trypanosome was also injected into a mare, in milk at the time, but without a foal. Microscopic examination of her milk subsequently revealed the presence of scanty trypanosomes in it on several occasions. Inoculation of the milk into rats also gave positive results.

From this series of experiments the author concludes that *T. brucei*, *T. eransi*, and "*T. lanfranchii*," are able to pass into the milk of an infected mother rat; and that *T. eransi* and "*T. lanfranchii*" can pass into the milk of the guinea-pig, as well as into that of the rat. "*T. lanfranchii*," moreover, can pass not only into the mother guinea-pig's milk, but also by this channel into the young which she suckles. Finally, this trypanosome can be demonstrated in the milk of an experimentally infected mare. The author recalls that NATTAN-LARRIER (*Rev. de Path. Comp.*, 1913, Dec., p. 282) had previously found that *Schizotrypanum cruzi* constantly passes into the milk, and *T. equiperdum* does so occasionally. He also refers to the observations of VELU and EYRAUD (1916), who state that a bitch infected with the horse trypanosome of Morocco communicated the infection to one of her pups in the process of lactation. [See this *Bulletin*, vol. 9, p. 34.]

[The foregoing experiments appear to prove, therefore, that trypanosomes are able to infect young animals by way of their mother's milk. They can be demonstrated sometimes in the milk of an infected mother—by direct microscopic examination, or by inoculation of the milk into susceptible animals.]

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The large proportion of positive results will be noted. It has been demonstrated, apparently, that—at least in certain hosts—*T. brucei*, *T. rhodesiense*, *T. gambiense*, "*T. lanfranchii*," and *T. eransi*, are all able to get into the milk of an infected animal, and there remaining in an infective condition. Infection on the offspring by imbibing the infected milk has been demonstrated in the case of *T. brucei*, *T. gambiense*, and "*T. lanfranchii*." It is conceivable that the milk was not the real route of infection of the young animals in these experiments. LANFRANCHI discusses other possibilities, but believes that his method of experimentation excluded them, or rendered an alternative interpretation highly improbable. It seems worthy of note that the trypanosomes studied were all pathogenic species in susceptible animals which cannot be regarded as their normal hosts. C. D.

MCDONALD (J. T.). **Treatment of Leprosy with the Dean Derivatives of Chaulmoogra Oil; Apparent Cure in 78 Cases.**—*Jl. Amer. Med. Assoc.* 1920. Nov. 27. Vol. 75. No. 22. pp. 1,483-1,487.

The Leprosy Investigation Station of the United States Public Health Service, established in Honolulu in 1905 and on Molokai, Hawaii, in 1906, and of which Dr. J. T. McDonald is the Director, has made many important contributions to the study of leprosy. Since 1918, the station has had the help of Dr. A. L. DEAN who following up the work of POWER on the chaulmoogric fatty acid series, has overcome the difficulty presented by the insolubility of these fractions of chaulmoogra oil by converting them into their ethyl esters, thus obtaining "thinly fluid oils readily absorbable from intramuscular injection." The fact that the Director of the Investigation Station is also Superintending Physician to the Kalihi Hospital has brought about a very satisfactory co-operation between the laboratory and the wards, and has greatly facilitated the work of research on these products. The standard preparation now used for weekly injection consists of the ethyl esters of the entire fatty acids of the whole oil with 2 per cent. of iodine by weight, chemically combined. The dosage begins with 1 c.c. as in intramuscular injection and is increased by 1 c.c. at every second or third injection, until from 2 to 6 c.c. is reached, according to the age and weight of the patient. For internal use, the mixed fatty acids, carrying 2·5 per cent. of iodine in chemical combination, are given in capsules, as it is thought that the fatty acids themselves are more likely to conform to the normal digestive processes than their ethyl esters. "We are therefore using by mouth a predigested oil or fat which is semi-solid at room temperature, and, in capsule, very easy to take. Its dosage begins with ¼ gm. per 100 lbs. of the patient's weight, three times a day, an hour or two after meals. This is gradually increased every two weeks until the maximum of 1 gm. per 100 lbs. of weight per dose is reached." Preparatory to injections, syringes, needles, etc., are sterilized for upwards of 30 minutes in the autoclave. An "all-glass" 20 c.c. Paris syringe with an antitoxin needle is used, the needle being inserted its full length into the muscles at a site in the upper and outer quarter of the gluteal region, after treatment of the skin by iodine. The author has only noted one abscess in a series of close on 7,000 injections by this method.

Some interesting references are made to leprosy fever, a phenomenon which appears to arise *only* in patients when under treatment, and which, while decidedly disagreeable, is found to subside in a short time and to be followed by decided amelioration in the patient's condition. The temperature rises and, within a few days, there appear on the face, forearms, or elsewhere, "a number of well-defined, circumscribed eruptions, usually circular, dull red, and raised from the level of the surrounding skin, thickest in the centre, and hot and evidently more or less tender to the touch." These elevations are not leprosy nodules, hardly ever contain bacilli, and tend to recede and disappear. Speaking of the diagnosis of leprosy, the author states that he has abandoned the opinion that "the microscope is the supreme agent of the final diagnosis of leprosy." He now considers that leprosy can be diagnosed clinically in some cases at times when the bacilli cannot be found, especially in nerve cases. Since October 1, 1918, no less than 78 patients have been discharged "on parole," apparently cured. These comprised 36 males and 42 females: the average age on admission was 23½ years, 49 were of the nodular type, 24 anaesthetic and 5 mixed. Of the total number, 55 were bacteriologically positive on admission and 23 were negative. S. L. C.

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i. PRICE (J. Dodds). **Antimony in Kala Azar.** *Brit. Med. Jl.* 1920. Sept. 18. pp. 453-454.

ii. ELWES (F. F.). **Antimony in Kala Azar.**—*Ibid.* 1920. Oct. 16. p. 610.

i. The writer of this letter draws attention to the superiority of the sodium salt of antimony tartarate over the potassium salt, which is much more toxic. The former is just as efficacious as the latter in its specific action in kala azar. He has personally given more than 3,000 intravenous injections of the drug and has watched the effect of over twice that number given by his assistants in Assam. The conclusion arrived at is that in this salt we possess the means of curing the vast majority of cases of kala azar, provided they are secured in the early stages of the disease. A young tea-planter who first showed the disease in September was perfectly fit by Christmas after having received 21 injections, representing 25 grains of the sodium salt. He was free from fever after the third injection. Beyond an occasional purge no other treatment was given. Another case, which did not commence treatment so early, was treated in India, but left for England before the treatment was complete. Though his fever had ceased and he had put on two stone in weight on the voyage home, he relapsed in England, where he was further treated by the writer. After the seventh injection his fever left him and he has remained free since. Between July 28 and August 12 injections were given every other day. The initial dose was 4 c.c., and this was increased by 1 c.c. at each injection, the strength of the solution being 1½ gr. in 10 c.c. of normal saline. The patient has now had 13 injections and is still under treatment. The object of referring to these two cases is to point out that with the sodium salt the fever in kala azar, even when the case is not of recent origin, is often lost quite as quickly as in the case successfully treated with acetyl-*p*-aminophenyl stibiate of soda referred to by Dr. MANSON-BAHR in the *Brit. Med. Jl.* of August 14, [see this *Bulletin*, Vol. 17.]

The writer prepares his own solutions of the drug, using normal saline and sterilising by placing the bottle in boiling water in a pan for 10 minutes. The solution can be repeatedly boiled without destroying its efficiency. Injections are given when the patient's stomach is empty, and he is made to lie down for half an hour afterwards. If there is the slightest tendency to cough after the injection the next dose is reduced by ½ cc. With the sodium salt, however, this is rarely necessary. The majority of the cases treated by the writer in Assam were cured after 30 gr. of the drug. A cure was considered as having taken place provided fever had been absent and the spleen markedly diminished in size and if no febrile reaction occurred after the injection of the drug. If parasites are still present there is usually a rise, sometimes only a slight one, after an injection, and it is an indication for further treatment.

ii The letter by ELWES contains some comments on the one just reviewed. The writer says that he has used a 2 per cent. solution of tartar emetic for kala azar and has carried the dose to 10 c.c. of the solution. When the dose has attained 6 c.c., severe cough, nausea and retching occur. By giving the dose on an empty stomach and keeping the patient absolutely still for 10 minutes after injection these symptoms are minimized. The writer thinks the use of the sodium salt as employed to such a large extent by DODDS PRICE is a distinct advance, but considers it possible that if a 2 per cent. solution were used instead of the 1½ grains in 10 c.c. as advised by that writer, unpleasant symptoms might also appear. However, the sodium salt seems to have a greater curative value than the potassium one, and hence can be used in smaller doses. He wishes to endorse the view that a cure cannot be said to have taken place unless there has been marked diminution in the size of the spleen. That sign he considers of the greatest importance. If the patient is discharged with only a slight reduction in the spleen, then relapse occurs, and subsequent treatment is very unpromising, whereas if the spleen has been considerably reduced it will continue to decrease in size after treatment has been suspended. When once treatment has been commenced it is most important to continue the injections until the apparent cure has been attained. C. M. W.

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CORRESPONDENCE.

SLEEPING SICKNESS.

The following extract from a letter, dated 7th May, 1921, from Dr. GEORGE PRENTICE, of Kasunga, N'gara, Nyasaland, may be of interest to Fellows :—

"You may be interested to know that with the cessation of free shooting in this area there is a very rapid advance of tsetse, and herds of cattle are going down before it, kraals being cleared; also that sleeping sickness has extended from the area where Dr. YORKE was working in Rhodesia, and has now crossed the Nyasaland border north of Fort Jamieson. My first case from this side of the border was probably a carried one, but now the disease seems to have got a hold in two villages.

"Four members of one family died and a child in another. The mother of that child is also affected, and is at present a patient here.

"If any of your maps show Mngwazu's village, on the Lingadzi, about 13° south, that is the principal village.

"These cases are occurring at an altitude of just 3,500 feet."

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TRANSACTIONS

OF THE

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

NOVEMBER 17TH AND DECEMBER 15TH, 1921.

VOLUME XV. NOS. 5 AND 6.

Proceedings of a Laboratory Meeting of the Society held at the London School of Tropical Medicine on Thursday, 17th November, 1921, at 8.15 p.m. In the absence of the President, the Chair was taken by Sir PERCY W. BASSETT-SMITH, K.C.B., C.M.G.

LABORATORY MEETING.

The Society is indebted to the Board of Management of the Seamen's Hospital for granting them facilities for holding the Meeting at the School of Tropical Medicine.

The following demonstrations were given :—

Drs. W. BROUGHTON-ALCOCK and J. GORDON THOMSON gave the following demonstration :—

1. *Eimeria oxyspora*, DOBELL, 1919.—Oocyst with a single mass of undifferentiated cytoplasm. A small amount of oocystic residuum was present at this stage.

2. *Eimeria oxyspora*, DOBELL, 1919.—In this oocyst the contents were divided up into four sporoblastic masses, No nuclear structure was visible at this stage, but there was a considerable amount of residual material in the oocyst.

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3. *Eimeria oxyspora*, DOBELL, 1919.—The four sporocysts had taken the characteristic shape and the cyst wall was formed. The sporozoites, however, were only developing, and appeared as oval bodies at either end of the sporocyst. In each sporocyst were numerous refractile bodies, but the nuclei could not be clearly made out.

4 and 5. *Eimeria oxyspora*, DOBELL, 1919. Two fully differentiated oocysts. Within the oocyst, in addition to the four sporocysts, there was a certain amount of residual material. The sporozoites were fully formed, and the nuclei could be made out in the majority of them, situated near the middle of the body, but rather near the posterior end. At the posterior ends of the sporozoites there could be made out the fusiform crystalline bodies.

6. *Embadomonas intestinalis*, WENYON and O'CONNOR, 1917.—Showed a typical flagellate form of this parasite, in which the nucleus, the two flagella, and the cystostome could be made out.

7. *Embadomonas intestinalis*, WENYON and O'CONNOR, 1917.—A typical cyst of the parasite, showing the carrot-shaped outline of the cytostome.

A series of photomicrographs and camera lucida drawings, illustrative of both the above-mentioned parasites, was also shown.

Professor R. T. LEIPER exhibited for the Department of Helminthology of the London School of Tropical Medicine a series of microscopical preparations which illustrated the tissue reactions of the host to helminth infections:—

(*a*) Cerebral sparganosis.

(*b*) Paragonimiasis.

(*c*) Clonorchiasis.

(*d*) Invasion of the bile ducts with *Ascaris lumbricoides*.

Major H. C. BROWN gave a demonstration of the results obtained in a method which, he hoped, would be useful in enriching the number of dysentery organisms in cultures from stools. He had been doing this work in conjunction with Mr. MUGGLETON, the Chief Laboratory Assistant of the Wellcome Bureau of Scientific Research, and as they had been unable to obtain cases of dysentery which were actually excreting the bacilli, they had to use saline suspension of a mixture of *B. coli* and

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dysentery bacilli. The maximum enrichment obtained in such mixtures was 11·8 times that in the control plate from the same mixture.

The method of enrichment consisted in adding a small quantity of homologous high titre serum to the mixture of organisms, and then plating out from the deposit after the agglutinated bacteria had had time to settle. The result of enrichment was shown in a MacConkey plate from an actual case of paratyphoid fever, in the plate from the saline suspension of stool, treated with serum, several hundred *B. paratyphosus* B colonies were seen, whereas in the control plate made from the same saline suspension, no pathogenic organisms were present.

This method differs from that advocated by CASTELLANI, in which the bacteria which it is required to isolate are sedimented by homologous sera, and the supernatant fluid is plated out. The preparation of the various sera used in this method is a laborious procedure, and it seems much more simple to use agglutinating sera for the pathogenic organism, and plate from the deposit.

The elementary fact that agglutinated organisms are still alive and capable of growth has been made use of in the present method.

The inactivated patient's serum, in the later stages of the diseases, might also be used in this way.

Dr. P. H. MANSON-BAHR exhibited a series of microscopic sections showing the various changes which take place in the gut as the result of bacillary dysentery. In the earlier stages the infection causes intense hyperæmia of the mucosa, followed later by necrosis, a process which may very occasionally involve the submucosa and muscular coats as well. In the repair of the damaged gut, sections demonstrated exfoliation of the necrotic layers, proliferation of macrophage cells in the submucosa, and attempts at regeneration by the formation of granulation tissue. Other sections illustrated submucous cysts and secondary infection of the gut wall with tubercle.

Charts and the clinical history of a case of liver abscess, which had burst into the right lung, were also exhibited. The patient had done very well on ipecacuanha and emetine; his temperature had become normal almost immediately, and he had put on over a stone in weight. The interesting point about the case is not the exceptional nature of the condition, but the fact that the sputum contained large numbers of yeast

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cells and mycelial threads. On cultivation yeasts of several varieties, as well as aspergilli, were isolated by Dr. DUNCAN. Immediately ipecacuanha was exhibited, expectoration ceased, and the yeast cells disappeared.

These yeast cells were obviously derived in this case from the abscess cavity, and were therefore to be regarded as a secondary infection, and not as a primary disease.

CASTELLANI has described cases of this nature as bronchomycosis, or bronchomoniliasis, on what appears to be very little evidence, and it is open to doubt, in view of the history of this case, whether the appearance of yeast fungi in the sputum is to be regarded as of any pathological or even diagnostic significance.

Drs. G. C. LOW and ELIZABETH O'DRISCOLL showed pathological specimens from a fatal case of blackwater fever; a supposed case of malaria which proved to be malignant endocarditis, and a series of specimens of trypanosomes from the cerebro-spinal fluid.

The blackwater fever patient had spent eighteen months in West Africa, where he suffered from slight attacks of malaria.

On the voyage home he was quite well and for the first part of his stay in London, when he suddenly developed fever with the passage of blackwater. He was taken into hospital at once and the usual treatment adopted.

On the second day the amount of urine sank to a few ounces and suppression threatened. By the use of saline intravenously, cupping the loins, etc., this was successfully combated and the amount of urine returned to normal. Though this was so, the patient did not do well; hæmorrhages began to appear from the mucous membranes—though the urine was now quite clear—and he gradually sank and died. During the progress of the case it was discovered that the blood gave a three plus Wassermann reaction. A good lot of the blood from the mouth came from an ulcer in the hard palate. The combination of the syphilitic infection raises some interesting points. Recently, many of the paroxysmal hæmoglobinurias have been shown to have strong positive Wassermann reactions, and it is possible that some of the many so-called blackwater fevers may be syphilitic in origin.

The organs of the malignant endocarditis case show what mistakes are

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being made with regard to the diagnosis of malaria in people who have been abroad: the patient had been treated for three years with quinine on the supposition that he was suffering from the latter disease. The autopsy showed typical malignant endocarditis, the enlarged spleen being due to septic infarcts. Dr. DUNCAN isolated a pure staphylococcus from the lesions. There was no evidence of any malarial infection.

The specimens of trypanosomes were taken from a new case of human trypanosomiasis just arrived from Nigeria. Though apparently only five months infected, a lumbar puncture showed that the cerebro-spinal already contained trypanosomes. Nervous symptoms of an obscure nature are present. The trypanosomes from the cerebro-spinal fluid are often degenerated and show peculiar forms. A series of these were shown.

Dr. ANDREW BALFOUR showed two poisonous sea fish from Mauritius.

The two curious fish exhibited in the next room are *Synanceia brachio* var. *verrucosa* and *Pterois volitans*. I owe them to the kindness of Sir HESKETH BELL, the Governor of Mauritius. They are known locally as laffes and are a source of danger to the bare-footed fisherman or bather. You will note their dorsal spines, large in the Synanceia, small in the Pterois. These communicate with poison glands which contain a powerful venom. In the case of the Synanceia, the grooves in the spines are closed by a membrane which converts the latter into sacs. This membrane must be ruptured before the venom is discharged.

Both these fish are passively defensive. They do not attack, but when trodden upon the spines penetrate the skin, break across, and the poison is ejected.

The ensuing symptoms are violent pain, spreading up the limb, great restlessness, and, in severe and untreated cases, death may result from cardiac failure. Sometimes there is local trouble in the form of suppuration, and even sloughing, while a secondary septicæmia may develop.

It is interesting to note that the Creole fishermen possess a cure which is said to be derived from a plant growing on the sea-shore. They make a poultice of it, and its application affords speedy relief. The secret is carefully guarded by certain families, and is handed down from father to son.

It would seem that here we have an opportunity for some useful and interesting research work.

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Dr. J. A. ARKWRIGHT and Mr. A. BACOT exhibited slides showing stages of Rickettsia in the sheep ked—*Melophagus ovinus.*

Rickettsia melophagi was first described by NÖLLER in 1917. He stated that it was constantly present in the alimentary canal of *Melophagus ovinus* and was hereditary, being passed on to successive generations of the insect. The originally described and generally recognised form of this Rickettsia is a minute dumb-bell shaped organism about 0·4 micron in diameter, which is found in very large numbers lying close to the epithelial cells of the fore and mid gut, adhering to the cilia of the epithelium, and consequently arranged in rows at right angles to the surface of the lining membrane. The same part of the gut which is inhabited by the Rickettsia usually contains also great numbers of *Crithidia melophagia* which, however, lie nearer the centre of the lumen.

During the past year we have examined a number of *M. ovinus*, and in the case of the adult insect have been able to confirm the constant occurrence of enormous numbers of the typical form of *R. melophagi* in the alimentary canal. We have also seen other forms in very large numbers, which we regard as the same organism under different conditions or in different phases of its existence. The films and sections have been stained by Giemsa. The following varieties of form have been found, and specimens were shown :—

(1) The very small form seen in masses in the fore part of the gut may be rod-shaped instead of dumb-bell shaped, though still of approximately the same size.

(2) Much larger forms, which are more in evidence in places where there are few typical Rickettsia or flagellates. They are seen especially in smears of the hind gut of the adult insect and in the alimentary canal of the embryo. These relatively large bodies vary in size from about 1 to 2·5 microns, or even more, in diameter, are round or oval in shape, and are usually deeply stained at the poles or on one side, but unstained in the centre, though some are stained throughout. These forms were at first regarded as an entirely different organism, but are in all probability *R. melophagi.*

(3) Intermediate forms between the minute and large forms are very numerous in some situations. These are short rods or oval forms, with poplar staining and clear centre. They vary very much in size and

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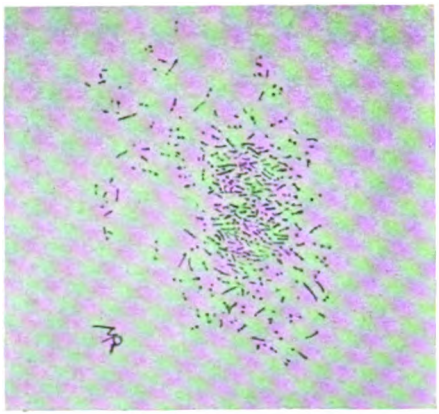

Fig. 1.

Portion of smear of gut of an adult "ked" *after feeding.*

× 1,500.

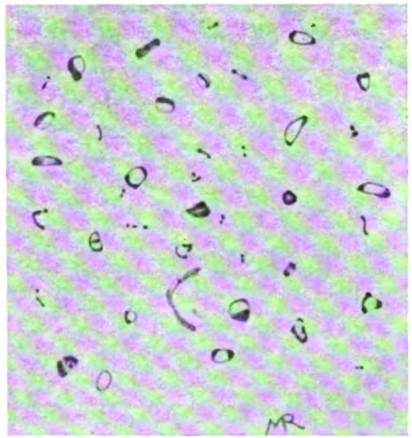

Fig. 2

Forms observed in smear of mid and hind gut of newly emerged *unfed* "ked."

× 1,500.

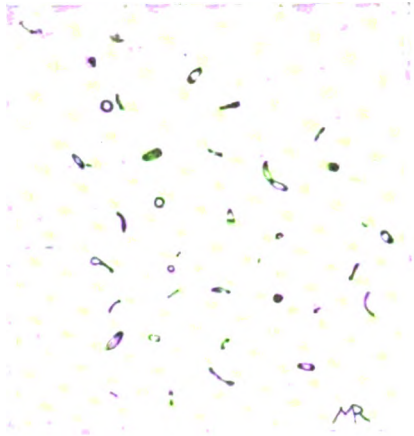

Fig. 3

Forms observed in smear of mid gut of newly emerged "ked."

× 1,500.

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shape, and appear to provide abundant links connecting the extreme forms.

These links were first observed and are perhaps most often found in the gut of the embryo dissected out of the puparium and of the newly-emerged adult insect; in the latter, beside a very varied assortment of the large and intermediate forms, small groups of minute Rickettsia are often seen.

The variety of shapes and appearances seen has a striking likeness to that exhibited by some bacteria under different conditions, and this similarity strongly suggests the bacterial nature of *R. melophagi.*

We are indebted to Miss RHODES for the extremely accurate drawings for the accompanying plate.

Dr. P. A. MAPLESTONE exhibited a smear stained with Leishmann, from a granulomatous swelling on the inside of the right cheek of a female Australian aborigine, *æt.* about 22.

The smear showed numerous organisms in heavily-infected large mononuclear cells, and also free (extracellular).

On account of the similarity of these organisms to those described by ARAGAO and VIANNA (Mem. de Instit. Ozwaldo Cruz, Vol. V), a diagnosis of ulcerating granuloma was made; the patient was treated with intravenous injections of tartar emetic, and it cleared up rapidly.

This case is interesting, as it is very rare, if not unique, to find the condition in the mouth, the genitals being practically always the part affected.

Professor WARRINGTON YORKE and Dr. J. W. S. MACFIE showed:—

I. A specimen of urine from a case of paroxysmal hæmoglobinuria indistinguishable from that passed during an attack of blackwater fever; and also specimens of blood from the same case, showing autolysis.

The autolytic reaction is obtained as follows:—A small quantity of blood is withdrawn and immediately placed upon ice for five minutes, and then in an incubator at 37° C. for one hour. On centrifugalisation the serum is found to be red. No hæmolysis occurs if the blood be not placed on ice before putting in the incubator, as the immune body present in the hæmoglobinuric blood only unites with the red cells at low temperature. The maximum degree of hæmolysis is obtained if the

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cooling be for from 5 to 7½ minutes; when the period of cooling is prolonged the degree of hæmolysis becomes less and less, until, if the cooling is prolonged for 60 minutes, the degree of hæmolysis is only about 10 per cent. of that obtained when cooling is for 5 minutes only. The explanation of this paradox is complicated and depends on complement splitting.

Tube 1.—Paroxysmal blood not cooled and put straight into incubator at 37° C. for 60 minutes. Result, no hæmolysis.

Tube 2.—Paroxysmal blood cooled for 5 minutes before putting into incubator for 60 minutes. Result, intense hæmolysis.

Tube 3.—Paroxysmal blood cooled for 1 hour before putting in incubator for 60 minutes. Result, slight hæmolysis.

N.B.—This reaction is not given by blood from blackwater fever patients.

II. Specimens illustrating the anatomy of *Oxyuris equi*, SCHRANK, 1788, RUDOLPHI, 1803, and *Enterobius vermicularis*, LINNÆUS, 1758, LEACH, 1853.

The worms exhibit the following striking differences in their internal anatomy:—

	OXYURIS EQUI.	*ENTEROBIUS VERMICULARIS.*
Ant. extremity ...	No cuticular flange.	Cuticular flange.
Mouth opening ...	Oval; surrounded by six equal inconspicuous lips.	Triradiate; surrounded by three relatively prominent lips.
Mouth cavity ...	Large, partly chitinised and provided with three prominent teeth in the female, and with numerous chitinised bristles in both sexes; these structures project into the mouth from the œsophageal funnel.	Minute and unarmed.
Œsophagus	Relatively short. Broad, cylindrical, ant. portion. Narrow, cylindrical, med. portion. Spherical, post. portion. No posterior œsophageal bulb.	Relatively long. Almost cylindrical, narrow, and dilating slightly posteriorly. Well-marked posterior œsophageal bulb.
Female genitalia ...	Large, single, sac-like uterus.	Two divergent uteri.
Eggs	Shells thick; large plug at one pole.	Shell not so thick; without any plug.

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III. A blood film of a rat showing a small spirochæte obtained by Dr. CORSON from a Gold Coast dog. The organism appears to be identical with *Sp. laverani*, BREINL and KINGHORN, 1906.

Professor BLACKLOCK and Professor YORKE showed blood films made from Sierra Leone natives containing ciliates resembling Nyctotherus. These organisms probably came from the water with which the films were washed.

Squadron-Leader H. E. WHITTINGHAM, R.A.F.M.S., gave a demonstration on a spirochæte isolated from cases of sandfly fever in Malta.

A spirochæte having morphological characters very similar to the *Leptospira icterohæmorrhagiæ* has been isolated from cases of sandfly fever occurring in Malta.

Briefly, the work was carried out in the following manner. A nominal roll of the Royal Air Force personnel at Calafrana, Malta, was prepared, showing the term of service out of England. the nature of any previous fever, and date of T.A.B. inoculation. From this roll it was easy to isolate all those who had not spent a summer overseas before. These men were inspected and had their temperature taken daily. Any who had fever were immediately admitted to sick-quarters. Those with a temperature of 99° F. were instructed to report at the laboratory a few hours later to have their temperature again recorded. In this way cases of fever were diagnosed in their early stage.

From previous work done by BIRT and others, it was proved that the blood of man was infective by direct transmission to other men only on the first day of disease. Thus it was decided to limit the blood-culture work to the first day of disease.

Every case admitted to the sick-quarters was carefully gone over, and only those showing the typical symptoms of sandfly fever were accepted for the investigation. In all, twenty-six cases of apparent sandfly fever were investigated. To aid in the differential diagnosis these cases had their blood examined daily for fourteen days for malaria parasites, total and differential leucocyte counts. Venupuncture was performed in every case on the first day of the fever. The blood collected was disposed of as follows:—

2 c.c.	was inoculated into	1 c.c. of Wenyon-Noguchi medium.
5 c.c.	,,	30 c.c. ,, ,,
5 c.c.	,,	30 c.c. of nutrient broth.
3 to 5 c.c.	,,	guinea-pigs intraperitoneally in 15 cases.

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The remaining blood was used to carry out the Wassermann test and agglutination test against *B. typhosus*, *B. paratyphosus* A and B, *B. enteritides*, GAERTNER, *B. dysenteriæ*, SHIGA and FLEXNER, *M. melitensis* and paramelitensis. On the 10th day of disease the blood was again collected for agglutination tests. The urine was examined daily, especially for albumen, bile and spirochætes.

Synopsis of results.

A. Blood cultures.
B. Inoculation of guinea-pigs with whole blood.
C. Inoculation of guinea-pigs with cultures of spirochætes.
D. Inoculation of man with cultures of spirochætes.
E. Blood counts.
F. Wassermann and agglutination tests.
G. Urine examination.

A. Blood Cultures.—Dark ground illumination of the primary blood cultures in the Wenyon-Noguchi medium revealed, after the 5th to 6th day of incubation at 27°C., the presence of spirochætal bodies in six cases. These spirochætes morphologically resembled leptospira. Three of these primary cultures were successfully subcultured. Unfortuately one of the subcultures was contaminated, and lost in transit from Malta to England.

B. Inoculation of guinea-pigs with whole blood.—Most of the guinea-pigs inoculated with the whole blood of sandfly fever cases showed a rise of temperature of about 2° F. for the first forty-eight hours after inoculation. Control pigs inoculated with blood from supposedly sandfly fever-free cases failed to show this rise. Two of the control pigs collapsed and died in the first twenty-four hours after inoculation. There was an epidemic of diarrhœa amongst the pigs—only a small number were really healthy before experimentation. Great difficulty was experienced in getting healthy pigs. All the pigs that died were examined, cultures were made of their heart blood, liver and kidneys, and the latter organs sectioned. In no case were spirochætes found. None of the pigs developed any symptoms of epidemic jaundice.

C. Inoculation of Guinea-pigs with Cultures of Spirochætes.—As the primary cultures might contain toxins as well as the spirochætes, they were not used for inoculation experiments on animals or man. Three guinea-pigs were inoculated intraperitoneally with primary subcultures, seven, eight and ten days old respectively. None of these pigs died

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or showed any symptoms of jaundice or hæmorrhage. Six guinea-pigs were inoculated with one week old secondary subcultures. One of these pigs died on the 10th day after inoculation. There was neither fever nor any symptoms of note. Attempts to isolate spirochætes from the heart blood, liver and kidneys failed. Six guinea-pigs were inoculated with four weeks' old primary subcultures. One of these pigs died on the 12th day after inoculation without symptoms of fever, hæmorrhage or jaundice, nor could spirochætes be recovered from its blood or organs.

D. Inoculation of Man with Cultures of Spirochætes.—Only three men have been inoculated up to date. Two in an area where sandfly fever occurs, and one in a sandfly fever free area.

Case 1 was inoculated with ½ c.c. of primary subculture (6 days old),
„ 2 „ „ „ ½ c.c. „ „ „ (6 „ „),
„ 3 „ „ „ 1 c.c. „ „ „ (6 weeks old),
and nine days later a further inoculation of 1 c.c. of a six-day old secondary subculture.

In none of the cases was there very definite fever. Case No. 1, had a temperature of 99° F. or over from the 6th to 8th day after inoculation. The highest point registered was 99·8° F. There was pain in the legs and suffusion of the conjunctivæ for several days in cases Nos. 1 and 2. Case No. 3 developed headache, anorexia, marked nausea, tendency to syncope, and pains in the calves on the 7th to 9th days after the first inoculation. The blood picture showed a distinct leucocytosis (15,000 per c.mm.). A similar result, with more pronounced symptoms, lasting for several days, followed the second inoculation.

On account of jaundice developing on the 11th day of disease in one of the sandfly fever cases under investigation in Malta it was considered inadvisable to do further inoculations in man.

E. The Blood Count.—The blood picture showed in practically all the cases a leucopenia (4,000 per c.mm.) for the first three to five days of the disease, followed by a leucocytosis shooting up to 15,000 to 20,000 per c.mm. on or about the 9th to 11th day of disease. This marked rise of the total white count on the 9th to 11th day coincides with a tendency for the fever to relapse in some of the cases. It should be noted that there were distinct relapses of fever in three cases. In one there was a relapse on the 11th day accompanied by jaundice, and a further relapse on the 25th day of disease unaccompanied by jaundice. The fever

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recurred on the 11th day in another case, and on the 12th day in a third case. Otherwise the cases selected gave the typical clinical picture of sandfly fever.

F. Wassermann and Agglutination Tests.—These tests were negative in all cases, due allowance being made for the protective inoculation with T.A.B. vaccine.

G. Urine Examination.—The urine in two cases showed a small amount of albumen; one of them also contained bile in a relapse on the 11th day of disease. No spirochætes were found in the urine.

Conclusions.—At present, owing to the inability to transmit the disease in typical form to man or animals, no claim is made to the specificity of the spirochæte isolated. Cross immunity experiments are in progress along lines kindly suggested by Lieut.-Col. H. MARRIAN PERRY, R.A.M.C. The above work was carried out by the Royal Air Force Sandfly Commission, which received very material help from many sources, including Squadron-Leader J. KELLY, M.C., P.M.O., R.A.,F. Malta; Major NEWMAN, R.A.M.C., Malta; Professor H. NOGUCHI, and various members of the R.A.M. College, Millbank, Lister Institute, Wellcome Bureau of Scientific Research, Medical Research Committee, and London School of Tropical Medicine.

Dr. A. CASTELLANI and Dr. DOUGLAS showed cultures of various fungi isolated from cases of tonsillomycosis—a condition which, in their experience, is much more frequently met with than is generally supposed. The fungi isolated by them belong to the genus Monilia, genus Hemispora and genus Diduim.

Dr. S. H. DAUKES exhibited a series of Indian cobras *(Naja tripudians)*, showing variations in colour markings.

Seven Indian cobras, received from Professor W. N. F. WOODLAND, were shown. All belonged to one species, *Naja tripudians.* The specimens showed a great diversity of colour and marking, but nothing in any way abnormal.

Dr. A. C. STEVENSON dealt with some microscopic queries:—

Six sections were shown in two groups of three each. In the first group two sections show a peculiar condition of a group of cells in the

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ventral wall of the stomach of the human louse. The cells in this group are distended by a vacuole which contains a mass of bodies of peculiar elongated shape, but with no definite structure. In the third section, bodies of the same appearance are seen in the ovum.

In the second group, two sections were shown of a group of bodies in the axillary fat of a white rat, bred in the laboratory and about a year old. These bodies consist of a thick capsule containing in some instances small uninucleate bodies. In amongst these are sections of curved bodies with thick walls. There are no signs of inflammatory reaction round the mass. The third section shows a piece of obviously vegetable tissue imbedded in the meninges of the brain. Here inflammatory reaction is seen. It is suggested that the brain lesion is primary, and the axillary mass is a growth of a mucor-like mould introduced with the primary lesion.

Dr. C. M. WENYON gave a demonstration on a simple culture medium which he had employed for the cultivation of leptospira and protozoa. He also showed a series of mice which had been permanently cured of a *Trypanosoma equiperdum* infection by means of "Bayer 205."

I. The medium is prepared as follows :—To 270 c.cm. of 0·85 per cent. sodium chloride solution (Ph. 7·6) are added 30 c.cm. of ordinary 2 per cent. bacteriological nutrient agar (Ph. 7·6). When mixture has taken place, 10 c.cm. are placed in each test tube. After autoclaving at 120°C. the tubes are cooled to 50°C., and into each tube are allowed to drop, from a rabbit's ear, 20 drops of blood. The tubes are not shaken nor mixed, and are incubated for twenty-four hours at 37° C. The medium is then ready for use. The blood is obtained from a rabbit by the paraffin method. The animal is enclosed in a box at one end of which is a round opening fitting the neck, through which the head projects. The ear is shaved over the marginal vein. The skin is sterilised with alcoholic iodine solution, and when dry is coated above, below and on the margin with hot melted paraffin, so that the area of operation is covered by a thin layer through which the vein is still visible. With a small sharp knife an incision is made in the marginal vein and the sterile blood allowed to drop from the paraffined margin of the ear into the tubes. After incubation of the blood agar tubes it will often be found that the blood has coagulated in a cylindrical column, leaving a clear agar medium around it.

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In this medium *Leptospira icterohæmorrhagica* grows readily, and sub-culture requires to be made every two or three weeks. It was on this medium that Squadron-Leader H. E. WHITTINGHAM isolated the leptospira from cases of sand-fly in Malta.

This medium has also been employed for the culture of certain intestinal protozoa. Thus from the case of *Embadomonas intestinalis* infection referred to by Drs. BROUGHTON-ALCOCK and J. G. THOMSON cultures were obtained. In this case the flagellates grew in association with numerous bacteria. The flagellate has now been carried through several sub-cultures, and it is evident that there will be no difficulty in maintaining it. In the culture both free and encysted forms occur.

In 1918, a species of Embadomonas, similar to *E. intestinalis*, was seen in a guinea-pig in Major URQUHART'S laboratory in Macedonia. Quite recently three guinea-pigs and a wild rat have been found infected with what appears to be the same Embadomonas in England, and cultures from all these animals have also been obtained on this medium. Similarly, a species of Trichomonas and a Blastocystis from the tortoise *Testudo radiata*, kindly supplied by Dr. MANSON-BAHR, is growing well in sub-culture. In the case of these intestinal protozoa, the cultures contain bacteria upon which the flagellates feed.

In the case of the leptospira, of course, no bacteria are present. In a similar manner, in pure culture both *Leishmania donovani* and *L. tropica*, as well as the Leptomonas of the flea and *T. lewisi* grow well in the medium. It has also been successfully used by Mr. C. A. HOARE for the culture of the trypanosome of the sheep and the flagellate from the sheep ked. The temperature of culture is 24° C. to 30° C.

The medium is a modification of NOGUCHI'S serum medium, but it has the advantage of being much more easily prepared, as it involves no separation of serum from the blood, which is dropped directly into the warm agar from the ear of the rabbit. In the preparation of N.N.N. medium for growth of leishmania or other flagellates, the same simple method of introducing the blood to the medium is employed.

A culture of *E. intestinalis* was first obtained by HOGUE (1921) in an egg medium, which has also given good results in the writer's hands. The egg medium is prepared as follows:—LOCKE'S solution is first made: sodium chloride, 1·8 grams; calcium chloride, 0·048 grams; potassium chloride, 0·084 grams; sodium bicarbonate, 0·04 grams; distilled water,

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200 c.cm.; to this solution is added one whole egg which has been well whipped. After thoroughly mixing, the solution is steamed for fifteen minutes over the water-bath, with constant stirring, and is then filtered through cotton-wool. About 5 c.cm. of the filtrate is then placed in each test tube. After autoclaving at 120°C. the tubes are ready for use.

II. A series of mice which had been cured of a very heavy infection of *T. equiperdum* over three months before were shown. The animals were treated by intravenous injection of "Bayer 205," the minimum dose required to effect a permanent cure being 0·003 grams of the drug for each kilogram of body weight. Over fifty mice had been treated with this or a larger dose, and there had been no single instance of relapse.

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Proceedings of a meeting of the Society held on Thursday, 15th December, 1921, at 8.15 p.m., at 11, Chandos Street, Cavendish Square, W. 1, preceded by a Demonstration at 7.45 of views, photographs and other objects of interest from Mauritius, by Dr. ANDREW BALFOUR, C.B., C.M.G., and Dr. D. E. ANDERSON.

Sir RONALD ROSS, K.C.B., K.C.M.G., F.R.S., in the Chair.

The CHAIRMAN: You will be pleased to learn that Professor MESNIL, of the Institut Pasteur, Paris, who is also one of our Honorary Fellows, has been elected to the French Academy, which is a very great honour. I think the Royal Society of Tropical Medicine would do well to send him our congratulations.

This was agreed to unanimously.

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A MEDICAL AND SANITARY SURVEY OF MAURITIUS: PAST, PRESENT AND FUTURE.

BY

ANDREW BALFOUR, C.B., C.M.G., M.D.,

Director-in-Chief, Wellcome Bureau of Scientific Research.

I have so many lantern slides to show that I do not intend to read any of the notes which you have already in your hands. The Government of Mauritius reported some time ago that the medical and sanitary conditions there were exceedingly bad, in fact worse than in any other British Dependency. As a result, the Colonial Office decided to send someone out to report on the question, and the lot happened to fall on me. My duties were to investigate, in the short period of six months, the whole conditions, medical and sanitary; to plan a scheme for the reorganisation of the Medical and Health Department, which had fallen into disrepute; and to advise as regards the allocation of a large sum, amounting to nearly a million sterling, which had been raised as a levy on sugar through the efforts of the Governor, Sir HESKETH BELL, for the purpose of carrying into effect necessary reforms and improvements.

I might begin with three famous words of Lord Macaulay, "Every schoolboy knows," and continue, where Mauritius is situated. I can only say that were I to do so I would be guilty of a misstatement. I have two sons—both schoolboys—and I found that neither of them had the vaguest idea as to what part of the world their father was proceeding. Furthermore, in the Municipal Chambers, Port Louis, one of the most treasured exhibits is an envelope which once contained a letter from Buckingham Palace, and which bears the legend, "Mauritius, West Indies." It is also true that a distinguished military officer accepted an appointment in Mauritius, believing it was in the West Indies, and was annoyed to find himself 600 odd miles east of Madagascar!

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I would not insult any Fellow of this Society by suggesting for a moment that he was not fully conversant with the geographical position of one of our more venerable colonies, but as memories are apt to be defective it may be well to furnish a few details regarding it.

Mauritius then is an island shaped like an oyster shell, and situated in the Indian Ocean between latitude 19°50' and 20°31' south and longitude 57°18' and 57°48' east of Greenwich. It is thus just within the tropic belt, but, owing to its southerly situation, its immediate surroundings and the influence of the Trade Winds, it by no means conforms to the customary idea of a tropical island. It is nearly 39 miles long and its greatest breadth is 29 miles. Its area closely corresponds to that of Surrey, being 716 square miles, while that of the home county is 758. Moreover, some parts of the island, notably Curepipe and Vacoas, towards the summit of the central plateau, recall in some measure certain features in the scenery of Surrey, and make it hard for the visitor to realise that he is in the southern tropics, many thousands of miles from the heights of Hindhead or the fir-bordered roads of the Farnham district.

So varied is the scenery, so bold are the mountain ranges, so large some of the rivers, that it is difficult to appreciate the fact that one is on a small island. It suggests a continent in miniature, yet it is impossible to go far in any direction without a view of the ocean, that glorious sapphire sea which circles the colony, and whose great breakers beat ceaselessly upon its coral reefs.

In a way Mauritius resembles some of the West Indian islands, yet it is a likeness with a difference, and I am not sure but that the difference is in favour of the old Isle de France, for it is a very lovely and attractive place and possesses advantages unknown in the Western Indies. Certainly, it is more favoured in the way of climate. The latter is mild and equable. According to the *Mauritius Almanac*—that storehouse of information annually compiled with great pains and much labour by Mr. Walter, the learned and energetic Director of the Observatory—the maximum shade temperature recorded at the Observatory, which is situated in the district of Pamplemousses, 178 feet above sea-level, has never exceeded 95°F., while in the highlands, as for example at the residential capital, Curepipe, 1,800 feet above the sea, it rarely reaches 80·4°F.

The relative humidity is high and hence during the summer the heat

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is at times oppressive, especially in the lowlands and more particularly in the capital, Port Louis, shut in as the latter is by an encircling belt of hills.

The only taste of the hot weather I experienced was during the month of April, and at that period Port Louis was unpleasant, though not unduly so. April, however, is not the worst month of the year.

From May to the end of September the climate is delightful, though a trifle cold and damp in the loftier parts of the highlands. Curepipe indeed is a very rainy place, so much so that its inhabitants are suspected of having webbed feet! Owing to the high relative humidity it is sometimes disagreeable in the cold weather, though the temperature never and nowhere falls below 44·6°F.

The rainfall varies a good deal in different parts of Mauritius, the mean annual fall ranging from below 29·9 inches on the north and west to 150·9 inches on the central plateau, where in years of maximum rainfall it exceeds 196·8 inches. Rain falls practically all the year round in the higher parts of the island. A distinct wet and dry season can only be differentiated in the coastal regions but, speaking generally, it may be said the rainy season begins in December, reaches a maximum in March and gradually decreases till June. There is a second maximum in August. The dry season occurs in October and November. During cyclonic disturbances, which are not uncommon, torrential rains may fall, a point of importance in connection with anti-malarial works. The physiographical features of the island will be pointed out on the screen. Here I would only say that Mauritius is of volcanic origin and its rocks are thus basaltic, consisting largely of trap, trachyte, and lavas. Some of the lavas are highly porous, and two varieties can be distinguished, *i.e.*, those containing large cavities and those with small pores filled with calcareous infiltrations.

This possibly has an important bearing on health conditions for, in many parts, the ground dries after rain with remarkable rapidity. Hence there are localities where surface water does not remain for any length of time, while outdoor recreation can be taken soon after heavy showers, a matter of considerable hygienic importance.

FOWLER, in his excellent *Malarial Investigations in Mauritius*, 1908, distinguishes the following five varieties of soil from which it will be

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seen that, as indicated, the above-mentioned condition is by no means universal:—

"(1) The loam variety, which is most common. This is porous and allows water to drain rapidly through it. It contains few springs. This is a healthy soil.

"(2) The red soil, coloured with iron oxide, a loose gravel mixed with clay. Underlying it are often found beds of pure clay, consequently water is liable to collect and stagnate in it. This soil bears an evil reputation as regards malaria. "Terre rouge" and "la fièvre" being almost coincident terms in the season.

"(3) The black clay, extending from a few inches to three feet in depth. A considerable part of Port Louis is built on this, and many other districts, where fever is very prevalent, are composed of the same.

"(4) Sheets of rock, evidently the remains of laval outflow, bare or covered with a shallow top-soil.

"(5) Sandy soil, chiefly along the coast. When mixed with disintegrated coral and decaying vegetable organic matter or rotting seaweed, it has originated outbreaks of several fever epidemics amongst the troops."

As is sometimes forgotten, it is essential to consider soil, sub-soil and deep-lying strata when discussing the effects of telluric conditions on the public health, at least in tropical countries where malaria is rife.

Pictures illustrative of malaria and anti-malarial work form perhaps the most important part of my lantern demonstration, hence it seems only fitting that attention should be directed to the historical aspect of the question, especially as malaria has played a very remarkable rôle in the recent annals of the island.

The work of Ross and Fowler is so well known that there is no need for me to recount how, possibly, the accidental introduction of *Anopheles costalis* into Mauritius, apparently somewhere about 1865, was the cause of a terrible outbreak of the disease. In Port Louis, out of a population of 80,000, 22,231 perished in the short space of thirteen months. Ever since that time malaria has had to be reckoned with in the colony. It has caused the loss of many lives and much treasure, and I firmly believe if Mauritius had not possessed in many parts so porous a soil and been blessed with such a remarkable winter climate it might

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easily have been ruined, as was ancient Greece, and as were large tracts of ancient Italy and Spain.

Mauritian doctors have studied and fought the fever; Mauritian entomologists have dealt with its vector; Ross has produced a classic on the subject, and, I am glad to say, conditions have greatly changed for the better.

I am of opinion that still greater changes can be produced, and that, even if the disease and its carrier cannot be utterly stamped out, they can both be so greatly reduced as to be almost negligible. I think and hope we stand at the commencement of a new era, where increased knowledge, sound administration, concerted action, and, above all, ample funds will in a few years revolutionise the situation.

Hence it seems well to look backward for a short space and realise what malaria meant, when, like a pestilence, it ravaged this island of the sea and when 234 people died of the fever in one day in the capital.

In the course of my reading I find the best and most graphic description of the great epidemic, which slew in all 31,920 persons, is to be found in a work bearing the curious title, *Sub-tropical Rambles in the Land of the Aphanapteryx*. This interesting book, which gives a good account of Mauritius in the late sixties of last century, was written by Colonel NICHOLAS PIKE, who was not only American Consul at Port Louis but was an ardent zoologist. Here is what he, as an eye-witness, recorded:—

"Those who inhabited Port Louis during the terrible mortality in 1867 and 1868 will never forget the sad spectacles the city presented daily. Fever! fever! was the only word on every lip, the only thought in every heart. Mourning and desolation everywhere. Scarcely a person visible that did not wear the garb of woe. Song and laughter had ceased.

"Port Louis was once remarkable for the number of pianos heard in every street in an evening, from the Erard's grand and semi-grand to the humblest cottage instrument.

"At this time it was literally 'The daughters of music were brought low, and the voice of mourning was heard in the streets.'

"Funeral trains were met at every corner. Relays of men were kept night and day digging the graves.

"The owners of undertakers' shops that sold mourning, and the

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apothecaries, must have made fortunes. The numerous druggists' shops were so crowded day and night, and so short of hands, that it was with difficulty medicine could be procured. Offices were opened in all directions for the distribution of food, medicine, or advice to the destitute; but all the efforts made by the municipality and private charities could not keep pace with the strident progress of the wretchedness and distress.

"There was no mistaking the appearance of one who had suffered: the pallid, drawn features, the skeleton, bloodless fingers, as if the bright life-stream had been dried out of them, and the slow dragging step marked but too plainly the victims.

"It was distressing to pass through the streets: in every corner was some poor creature, suddenly struck down, and crouching on the ground to die.

"In the outskirts of the city and country roads the victims were so numerous that the police and sanitary committees were insufficient to succour half the poor wretches, and many died by the roadsides before help could be brought to them.

"Near Roche Bois I have seen them lying in groups, dying and dead. Not a house, within a radius of half a mile from the one I then occupied, had a living person in it, except at a shop belonging to three Chinamen, two of whom died later.

"In many cases, as soon as a Malabar got the fever, he would hasten to his house and shut himself in to die; for such was the fear of it, to be attacked was the tocsin of death to him.

"I visited many families, and the scenes I witnessed will never be effaced from my memory. A poor Indian, whom I had cured for the time being, came and entreated for help to a comrade. It was night, and I was tired and had gone to bed; but the poor fellow begged so hard that I dressed and went with him. After a long walk we came to a hut, and as I approached I heard groans and lamentations. When I entered the scene baffled all description. A small cocoa-nut oil lamp dimly lighted the interior, adding horror to the scene.

"It was inhabited by a man and his wife, with a number of children. The mother lay dead in the middle of the hut, the man hanging over her in an agony of grief. Her baby, still living, was clasped to her heart,

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and seeking to draw its life-sustenance from her cold breast. The other children were all stricken with the fever, and in its last stages, past human help. Of course all I could I did, but help had come too late to do little more than assist in their burial.

"One dreaded to ask the news, as one was quite sure to hear of some friend ill, dying or dead, and often buried before you knew of it. Parents had to rise from their sick beds to nurse their children, and these again had to drag their weary limbs to follow a beloved parent to the tomb, though frequently too weak even to do that.

"No change of weather seemed to arrest the plague. Intense heat or cold, heavy rains or dry, mild calm days, or sharp breezes, all were alike fatal. The brightest morn brought no more hope than the wildest night.

"For months the death-rate in the city alone averaged nearly 200 *per diem*. In every street could be seen the mourning weeds outside the doors where death had struck his victim and this was often the first intelligence you had of the loss of dear friends—no time for ceremony then. May I never witness again the sad sight of those incessant funerals, slowly wending along from morn till night.

"Here a group of Malabars bearing along some poor fellow, preceded by a priest muttering a prayer, and followed by a few women bearing a copper dish of rice and fruit, and a jug of water, to place on his grave.

"There comes a slow and stately train with black-plumed hearse, and a long line of carriages behind it—one of the rich and respected of the land; anon, a little simple bier, bearing a baby's coffin covered with a simple white muslin pall and wreath, with perhaps only the father and nurse as mourners; then a white-covered hearse, its white plumes and the horses' sweeping trappings showing that some fair girl had been cut off in early womanhood.

"Occasionally would pass a Chinese funeral, the bier supported by stout Malagash bearers, in their long black gowns and flowing weepers looking as stolid as if of stone; a few carrioles following with Chinamen in them, and a person always preceding it, scattering pieces of paper about three inches square, often gilt or silvered, all along the road, to scare away evil spirits and prevent their following the corpse to its last resting place.

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LINES ON THE CEMETERY AT BOIS MARCHAND.

They lie in thousands side by side,
  On that wild desert plain;
The loved, the cherished, nameless there,
  By raging fever slain.

In tombs of their ancestral dead
  Their bones may never lie;
No marble records shield their graves
  Beneath that torrid sky.

O'er that blent mass of human clay
  No mourners bend in tears;
No wreaths, no votive offerings there,
  Though the loss will be felt for years.

For there the grey-haired grandsire lies,
  With the darlings he loved so well;
And there the bride of a few short hours—
  Of such griefs what tongue may tell?

The mother with her first-born babe,
  The father in manhood's pride;
The fairest and best were swept away—
  Our friends so trusted and tried.

Long, long will the "Gem of the Ocean" rue
  The fever fiend's deadly rage;
For sadder sights than its shores have seen
  Rest not in History's page."

Let us hope he was a better naturalist than he was a poet. The verses which I have read to you do greater credit to his heart than to his head, for neither his scansion nor his imagery is above criticism. Still they must have a place in the literature of malaria.

I found his succeeding remarks on the etiology of the disease so curious and interesting that I have transcribed them. Remember he was working in the dark and that he was not a medical man. In any case the medical men of the period had very similar views and the authority he quotes in support of his argument was, I believe, a doctor. Thanks to LAVERAN, MANSON, ROSS, GRASSI and many others, we know better nowadays and can explain, for example, why people living near marshes at a high altitude, such as Mare aux Vacoas and Bassin Blanc, escaped the fever.

"Everywhere one heard the questions, 'What have been the predisposing causes of the epidemic? and what are the existing causes of its long continuance?'

"Whatever may be the difference in opinion as to the origin and modes of propagation, all agree that certain states of the air favour the

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disposition of the body to receive intermittent and remittent fevers, and rivet them into the constitution, which baffle all attempts at complete cure, and induce a tendency to relapse from apparently slight causes.

"The concurrence of cold with a moist atmosphere; heavy rains after long dry weather; weakness of body, whether owing to poor and unwholesome diet, fatigue, severe evacuations, or previous diseases; anxiety of mind; inactivity, intemperance, or restlessness, all increase susceptibility; while hope, confidence, cheerfulness, whatever can excite mental energy, lessen it.

"Differing as intermittent and remittent fevers do in many points, particularly in their rate of mortality, they yet agree in their origin as occasioned by effluvia emanating from putrid, stagnant waters, swampy low grounds, and animal matter. It is found in the tropics that malarious diseases are most common in the seasons succeeding heavy rains, when the temperature is high, and where the surrounding country abounds with jungles, and insect life is rife.

"It is now well ascertained that gases, emanating from decomposed vegetable and animal matter, generate confervaceous as well as diatomaceous plants, such as *Tetraspora nostoc* and all the genera Agaria.

"These cryptogams are never found in *dry* warm situations, but where it is *damp* and warm, and they develop themselves especially where organic matter is in a state of putrefaction.

"Some of these plants live on the surface of stagnant waters, but very many on the surface of low lands. Others are parasites on plants, which they destroy, as is shown in the diseases of the vine and potato.

"In *Oidium tuckeri* the spores are so small that EHRENBURG, the great microscopist, was scarcely able to detect the form of a thousand of them, grouped together, with the highest power of the microscope. Our knowledge of the elementary structure of organisms is exclusively based on microscopic discoveries, and modern physiology is the result. Organic chemistry has materially participated in the development, but the microscope excels the chemical re-agents in practical usefulness, both as to precision and facility, and has firmly established its superiority in histology and physiology.

"Through the medium of these fundamental branches it has benefited medical science at large, and of late has lent material aid as well to diagnosis.

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"Sometimes it may delude and give rise to erroneous inferences, but the chances are in such a case it is the performer or an imperfect instrument that is at fault.

"It more frequently reveals the true state of elementary structure and its deviation from the normal state, and thus aids as well as corrects pathological knowledge.

"In a long-continued series of observations, in cases of persons who have died of fever, when particular organs and their secretions were submitted to minute microscopic examination, it was almost invariably found that the membrane lining the stomach was covered with a multitude of very minute plants, closely resembling the Alga, *Cryptococcus cerevisiæ*. These parasites often covered the whole intestinal tract; some were perceptible on the surface of the lungs, and some could be detected in the blood. In the latter, it sometimes happened that there were epithelial cells, apparently containing fatty and pigment molecules.

"On living patients, in the advanced stage of the fever, they may be detected, by one well acquainted with the microscope, in the substance which is formed at the corners of the mouth and eyes.

"Some of the parasites appear quite hollow, others contain nuclei and spores, and others show cell articulations.

"In the secretions of entirely healthy persons they cannot be detected.

"Water taken up in well-cleaned basins out of some isolated pools at Grand River, or where its waters mingle with the sea, and subjected to the rays of the sun till stagnant, developed a green superficial film. Under the microscope this film showed plants so nearly related in shape and structure to those in and on the different organs of the human body, that there is no doubt of their being of the same genera, and it is equally certain that they were exciting causes of the epidemic.

"Myriads of these plants were generated all over the island; and when matured, the spores became free, and were taken up by the wind and carried from place to place. These spores were thus inhaled, and if the stomach was not in a morbid condition, they would pass out without effecting or undergoing any change, or be destroyed by the gastric juices; if, on the contrary, it contained material highly susceptible of fermentation (which the universal rice and vegetable diet here tends to), the spores would germinate and grow, produce inflammation, and fever readily ensued.

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"Unlike phanerogamous plants, which absorb carbonic acid gas, depositing the carbon in the plant, and throwing off the oxygen into the atmosphere, all the lower class of cryptogams absorb oxygen, and consequently give out carbon, thus vitiating the atmosphere we breathe.

"It may not be out of place to mention here that the physiological effects of the cinchona bark, and its alkaloids, on vegetables, animals and men, should be borne in mind, as the connection of these effects with the therapeutical influence of the bark in fever was until lately inexplicable.

"DECONDOLLE states that leaves of plants immersed in an infusion of pale bark were dried in twenty-four hours; and others, plunged into a solution of quinia water, presented evidences of contraction in from six to eight hours.

"It is evident, therefore, why this remedy, when used in malarious fevers, acts so beneficially. By this contraction, or process of withering, it destroys the fast growth of the poisonous fungoids in the system, and, if there is no complication, eradicates the disease.

"This fever is only contagious under certain circumstances. Knowing how it is germinated, it will be readily perceived that when a person is attacked with it, in damp unventilated places, it follows as a matter of course, that from the moment the spores in or on the patient become free, all the inmates of the same place, and especially when they are crowded together and filthy in their habits, will be subjected to the disease; and in so far only is it communicable.

"Since writing the above, I met with an interesting article on 'The Causes of Ague,' and as it bore so forcibly on what I had written, I transcribe some of its paragraphs at length.

"'The fertile source of desolation and disease consists of incalculable myriads of microscopic cells, suspended in the atmosphere over waste, marshy, and fen districts. They are minute oblong cells, single or aggregate, and having a distinct nucleus, with a clear interspace, apparently empty, between it and the cell wall. They are of the algoid type, strongly resembling the Palmella, and are consequently amongst the very lowest organisms known. Sometimes several of these cells or spores are contained in an outer cell or wall, or delicate investing membrane, to form a plant.

"'Of these "ague plants" is formed the greyish film wherever damp, black earth is turned up and exposed to the sun. These spores or minute

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seeds (germinating cells) rise into the air, carrying pestilence with them. There are several species of the "ague plant," which have been called Gemiasma, from the Greek for earth, and the word miasma. The white, and a yellowish-green variety, occur usually in a non-calcareous soil, and produce agues of but slight intensity, and are the only ones known in England. There are also red, green, and lead-coloured "ague plants," and one singular species, the "*Gemiasma protuberosa*," which has larger spores, "and consists of groups of jelly-like protuberances."

"'These latter kind habitually occur in rich calcareous soils, and produce fevers of a dangerous and congestive character.

"'The cells with their spores produce visible incrustations of mould on the surface of recently exposed marsh earth. The danger from these growths is greatest in a hot dry season following a wet one. The wetter and hotter, the worse for man, and the better for malaria.

"'The marsh demon is verily "the pestilence that walketh in darkness." It seems almost certain that the spores of the "ague plant" only rise with the evening dews. Microscopically tested, the day air is free from those organisms.

"'In different parts of the world these cryptogamic spores rise in the night mists to definite heights.

"'In the United States they seldom rise more than from thirty-five to sixty-five feet above the low levels; in England not more than from fifteen to thirty feet.

"'These spores are found throughout these vapours, but do not extend beyond them, and are found in the greatest abundance in their upper strata.

"'Intermittent or ague fever has actually been produced in men by causing them to inhale the spores of these algæ.

"'It has long been known that malaria is movable by the wind, the spores of the "ague plants" having risen and become entangled in the mist; spores, mist and all are blown along together, far perhaps from the place of germination.

"'This fact admits of considerable practical application in tropical climates, where the wind usually blows for a long time from the same quarter. It is easy to see how a volume of vapour or fog, laden with its deadly burden of poison-cells, may roll up and hang suspended on the side of a hill, towards which a wind blows across an adjacent marsh.

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"'Instances have occurred where the poisonous vapour has been blown over a hill, and deposited on the other side, to the unmitigated disgust of the inhabitants, who imagined themselves secure from their pestilential neighbour.'

"The above article applies particularly to Mauritius, as I shall endeavour to show by a slight description of the numerous hot beds of the 'ague plants' with which the island abounds at the present day.

"I will begin with the very focus of malaria, Port Louis, and mention a few of the numerous causes of infection in that city alone.

"The foul streams flowing through it in all directions are, two-thirds of the year, almost stagnant; the other third they are swollen by the torrential rains, and bear along masses of vegetable and animal matter from the hills and Indian camps, which, as the waters subside, lie festering in the sun, poisoning the atmosphere.

"The emanations from the open drains, the imperfect drainage of the houses, and the defective method of disinfecting the night-soil, load the air with mephitic vapours.

"The gradual filling-up of the east end of the harbour from the mud constantly pouring into it, and the tide not being strong enough to wash away the impurities that lodge in the muddy bottom, adds its quota to the malaria.

"The low shores to the west of the city are only covered at high tides, and are strewn with decayed sea-weed and filth, washed in from the shipping.

"Between Port Louis and Port William lies a swamp, that receives into its rank vegetation the streams that flow from the cemeteries and another swampy land at the back of them. These cemeteries contain in themselves a very sufficient cause of malaria. The emanations from them are at times most deadly, owing to the circumstances that the dead are not interred deeply enough, and the loose earth and coral which cover them permit the escape of the gases evolved by their decomposition.

"It has been frequently remarked that the health of the city has invariably suffered from the miasma from its western side that has been blown over it.

"Between Port Louis and Grand River are low lands, prolific in the germs of malaria. After heavy rain the Grand River swells, and receives

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into its floods filth of every kind, which is swept down, or deposited all over its course, or left in pools to decompose in the sun.

"Near its entrance to the sea, where the waste water spreads out all over the wide embouchure made by the torrents of ages, and the rapidity of the flood abates, it has not power to sweep away all the débris, and part always remains filling up its channel, and impeding its proper egress to the ocean. The whole of this district, and the neighbouring one of Petite Rivière, lie low.

"Just before the fever broke out at Petite Rivière, there was a large camp of Indians located there, reeking with indescribable filth. The huts crowded to excess, men and beasts herded together; and with the ordinary dirty habits of the men, and the scarcity of water in that district during the severe drought at the end of 1866, the wonder is the fever left any of them alive.

"Very few indeed were spared; and it is a melancholy sight, at the end of 1867, to pass by the camp. Here and there you meet a poor squalid wretch, and a few weakly children; but nearly all the huts were destroyed that had contained whole families, now swept away, and the few that remained, shut up—it looked like a city of the dead, after teeming with busy noisy life as it did some months previously.

"The ill effects of allowing the Indians to wash their clothes and bathe in the running streams, thus polluting the waters in their whole course, were well shown during the epidemic, as death with unceasing energy stalked amongst those who lived near such waters, and used them unfiltered.

"The marshes of Pamplemousses and the West Savanne; the moist lowlands of Petite Savanne; the shallow tidal lagoons west of Black River; and all the low coast-line receiving the drainage from the central watershed, gave out their poisonous exhalations.

"During 1867 some parts of the island entirely escaped; most probably lying above the spore level, or fever line, which I see Dr. REID, the chief medical officer, places at 600 feet above the level of the sea.

"In his report on the fever, he mentions a curious fact about the spread of the fever into a section of Black River and Savanne, always known as the healthiest part of Mauritius. Between these districts and the infected ones lies a barrier of forests and woody elevations. He writes, 'During the first week of January, 1868, occurred a hurricane, the

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main force of which was from the south-east and east. in that extremity of the island sufficiently strong to spoil the forests of their leaves, and make gaps in this barrier of wooded highlands, and thus carried in the fever germs from the part of Black River, where fever was rife, to the hitherto healthy inhabitants.'

"I could add greatly to these details; but those I have mentioned are sufficient to show that, with all these existing powerful agents to malaria lying dormant, and so many spore-beds waiting for peculiar atmospheric influences to set them free, the heavy rains, and then excessive drought followed by hurricanes, would act on them with fatal certainty, and thus strike the whole island with this terrible plague, converting, for the time being, the once 'Gem of the Ocean' into a very pest-house.

"Dr. REID also mentions in the same report a circumstance which would seem to corroborate the fact alluded to on p. 98, as to the effects of the cinchona, as stated by DECANDOLLE.

"He says, 'The waters of the few remaining woodland marshes of Mauritius, the Mare aux Vacoas, aux Joncs, and Bassin Blanc, are deeply tinged and impregnated with tannin and resinous matters, and the inhabitants around and near these marshes entirely escaped the fever.'

"He suggests, and very properly, 'May not this exemption be due to the tannin, and other products of the débris of the surrounding forest, being present in these localities, and preventing the fever germs from flourishing therein? May not the removal from the humus and marshes of the lowlands of similar substances, which they received when the island was well wooded, have been one of the changes which prepared them for the reception and development of amalarious germs?'

"I do not doubt it; and it is most likely the withering effects of the different barks in solution, that prevented the germination of the fever-spores in those marshes, and caused the consequent immunity of the neighbouring inhabitants from the disease."

I do not think it is a waste of the Society's valuable time to see how men groped in darkness and how they tried to explain phenomena which nowadays we fully understand. In respect of certain diseases we stand where they stood as regards malaria, and a consideration of their errors and of their strivings towards the light may aid us to avoid pitfalls and guide us to a correct explanation of the problems with which we are faced.

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Some of these exist to-day in Mauritius, which we shall now study from various aspects of hygiene by means of lantern slides prepared from photographs I took during six months' inspection and advisory work in this famous and unique little colony, which the 1921 census has shown to possess a population of 376,108, of whom the great majority are Indians.

Over 120 lantern slides were exhibited illustrative of the following subjects:—

1. Geography of Mauritius.
2. Scenery of the island.
3. Malaria and antimalarial work.
4. Mosquitoes other than anophelines and antimosquito work.
5. Water supplies.
6. Washing arrangements.
7. Waste-water disposal.
8. Food.
9. Conservancy.
10. Refuse and refuse disposal.
11. Plague.
12. Ankylostomiasis.
13. Buildings of various kinds and sanitary and insanitary premises.

A selection of the above is here reproduced to illustrate the more important points considered:—

Geography.—A map of Mauritius was exhibited and the chief physiographical features of the island explained. Special attention was directed to the intensely malarious Black River district, an area of comparatively level land lying between high hill ranges and the sea.

Scenery.—Various views were shown, especially of the waterfalls, for which Mauritius is famous. Pictures of certain of the hill ranges and the larger rivers were also exhibited, together with illustrations of the beautiful coast scenery. Slides of Port Louis and its harbour formed a feature of this section, and an old plan of the capital showed the general layout of the town and its immediate surroundings Stress was laid on the Port's similarity in many respects to Port of Spain, Trinidad, and, from the point of view of plague, attention was directed to the fact that large ocean-going ships lie out in the harbour and do not tie up to wharves or quays.

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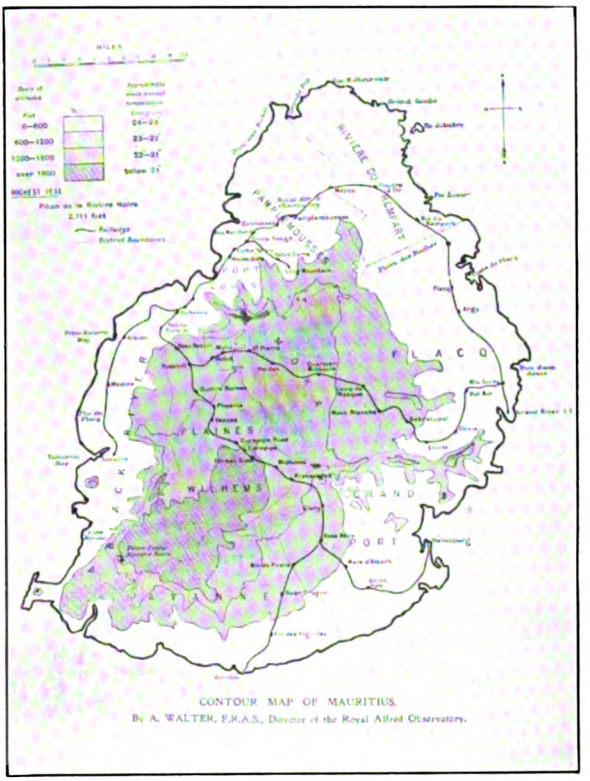
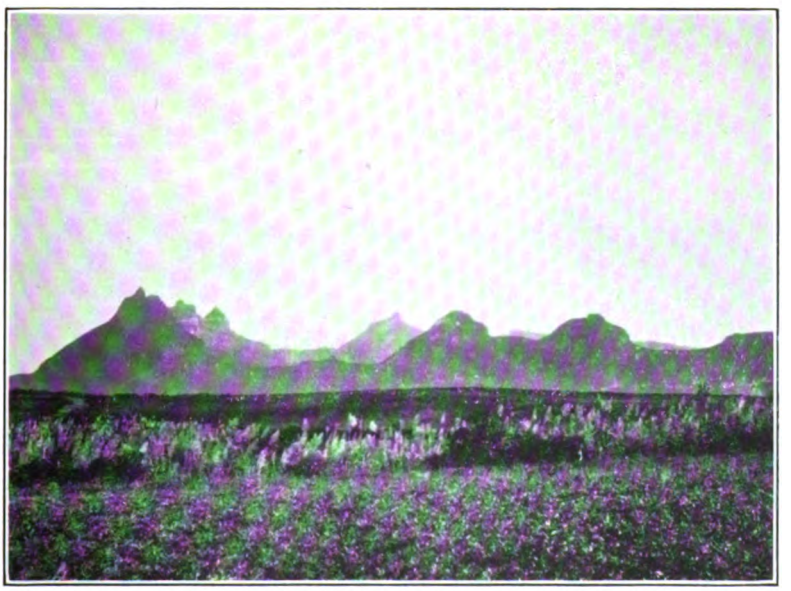


CONTOUR MAP OF MAURITIUS.
By A. WALTER, F.R.A.S., Director of the Royal Alfred Observatory.

1.—The island is well supplied with both roads and railways.



2.—The Moka Hill Range from Pamplemousses, showing the lofty summits of Pieter Both on the left and the Pouce in the centre.

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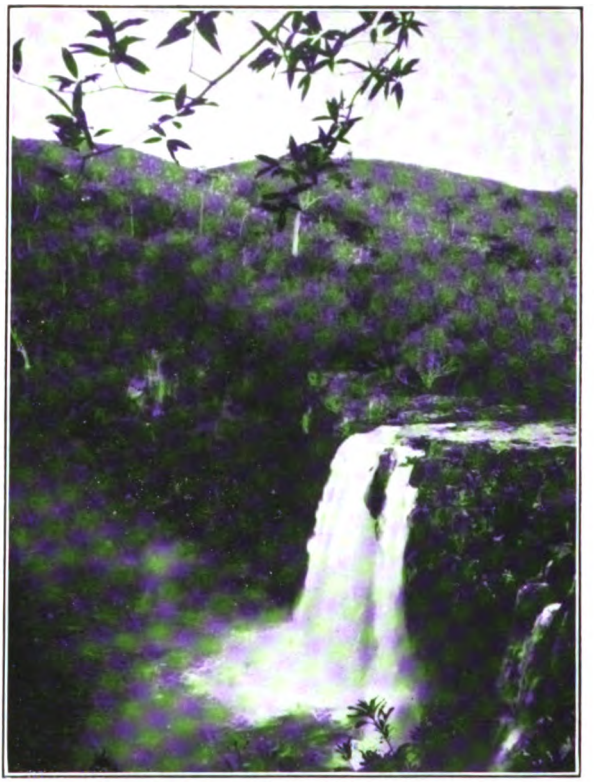
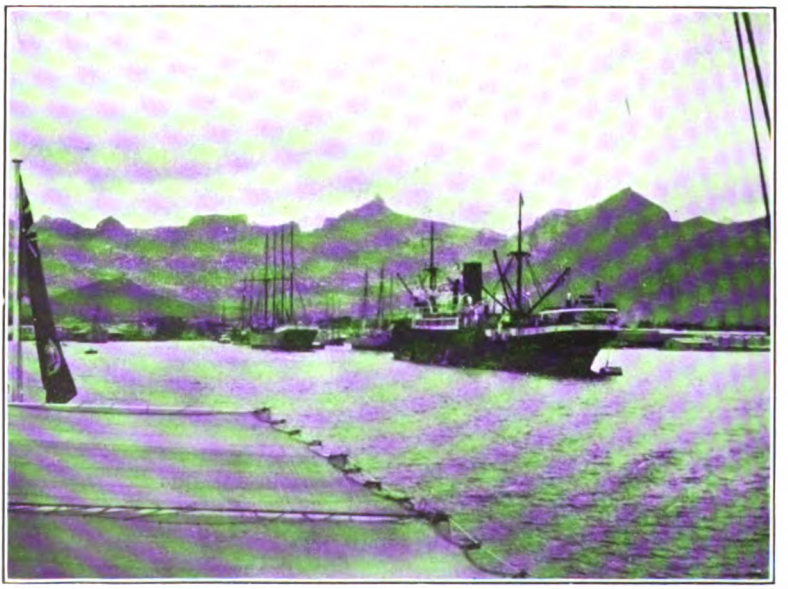


3.—Dia Mamour Falls, Grand River, S.E.



4.—View of Port Louis Harbour and Town from the sea, showing the Moka Range in the background with the Pouce Mountain in the centre.

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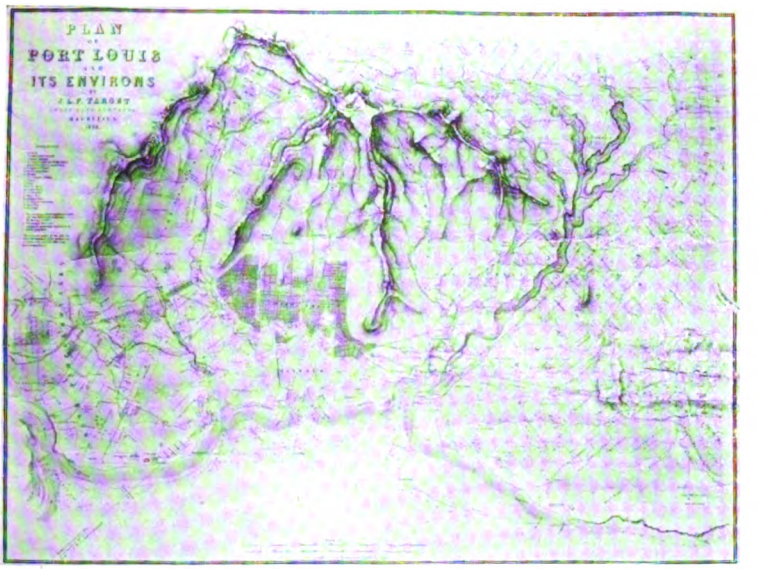
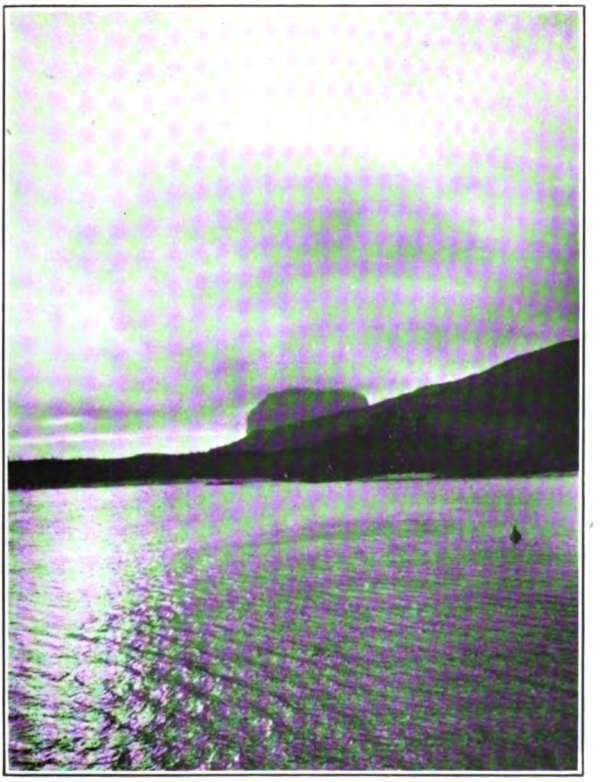


5.—Old map of Port Louis, showing the relations of the town to the surrounding hill ranges. The town is now not so extensive as here indicated, a considerable portion of it having been destroyed by the great hurricane of 1892, and never having been rebuilt.



6.—View across Baie du Cap. The mass of Morne Brabant in the background

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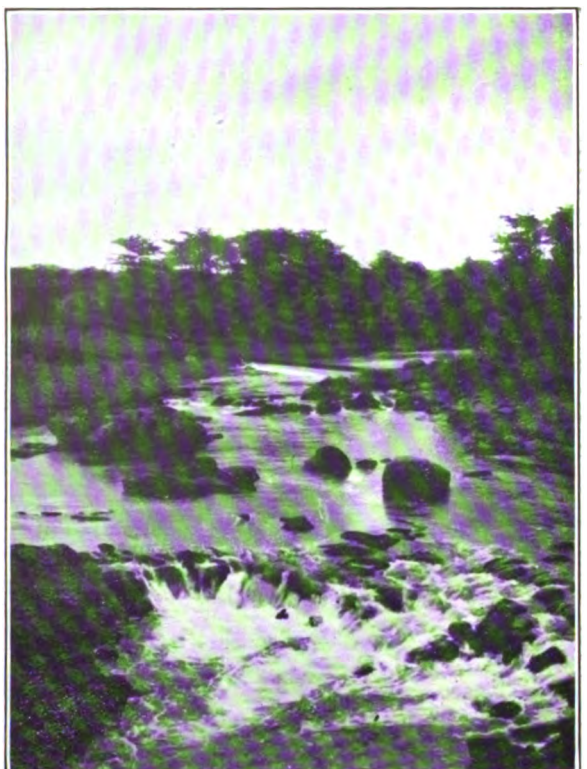

7—View of Grand River, S E

8.—Monument in the Bois Marchand Cemetery to a large number of the men of the Mauritian Police Force who died of malaria during the great epidemic.

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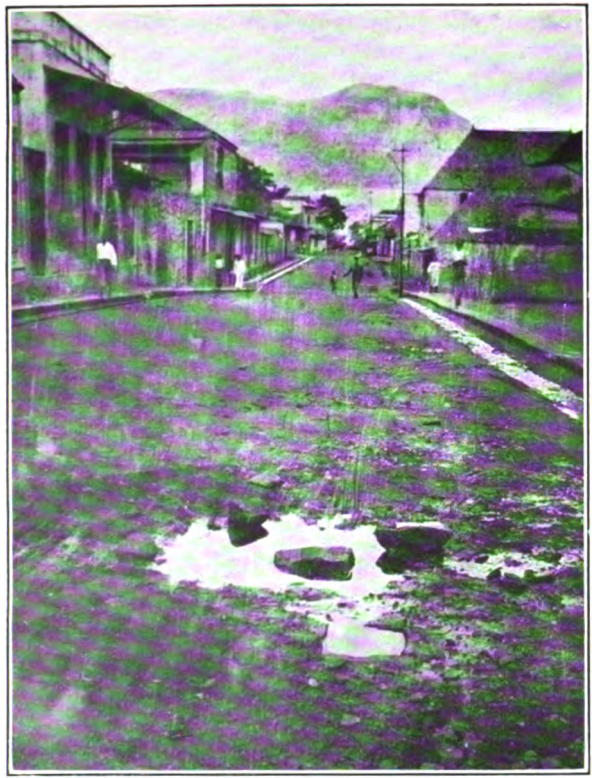
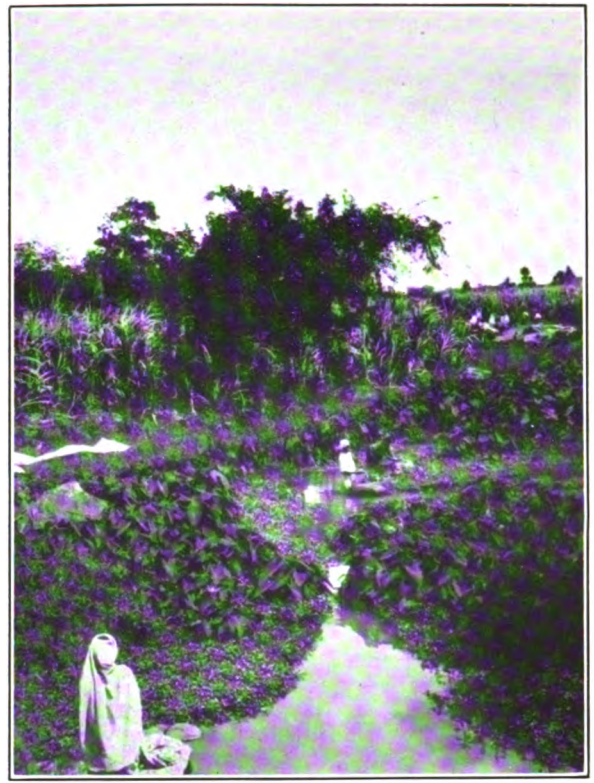


9.—A leaking "Regard" in one of the Port Louis streets—breeding-place for *Anopheles costalis*.



10.—An overgrown and neglected watercourse on a sugar estate, Pamplemousses.

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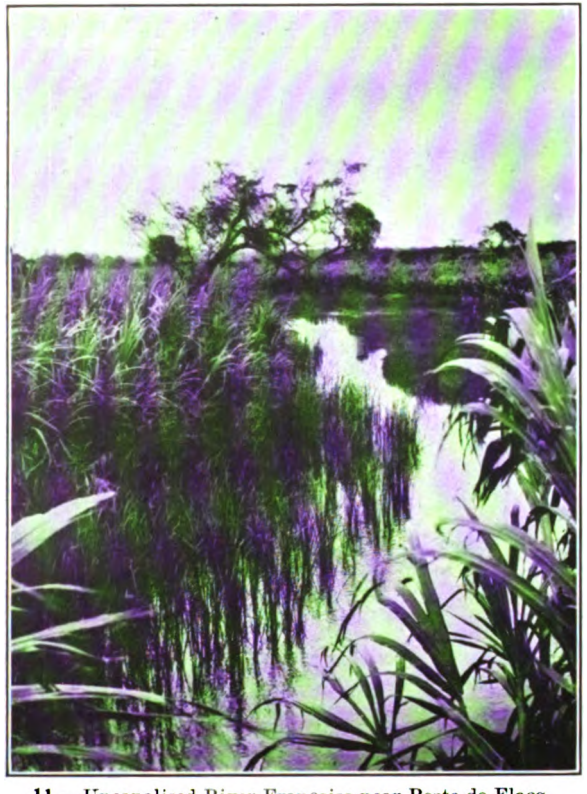
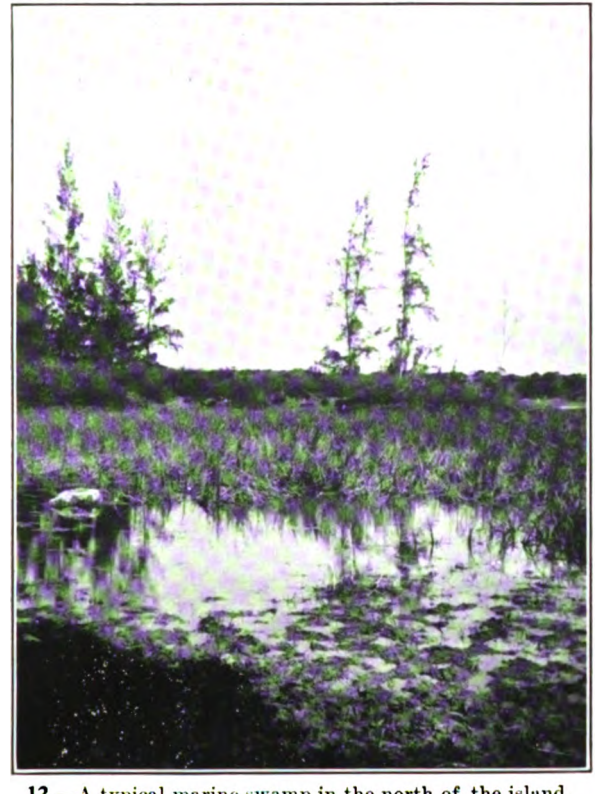


11.—Uncanalised River Françoise near Poste de Flacq.



12.—A typical marine swamp in the north of the island.

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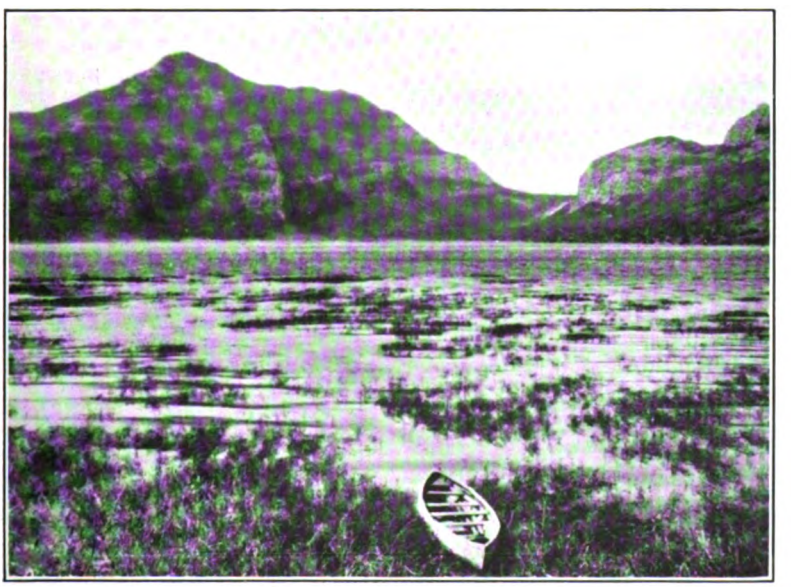
13.—An inland swamp at La Lucie, Fracq.



14.—La Ferme Reservoir. The water is at a comparatively low level and convenient breeding-places for *A. costalis* are provided.

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15.—Portion of Black River at the time of Ross' and Fowler's visit, 1908.

16.—Same portion of Black River at the present time.

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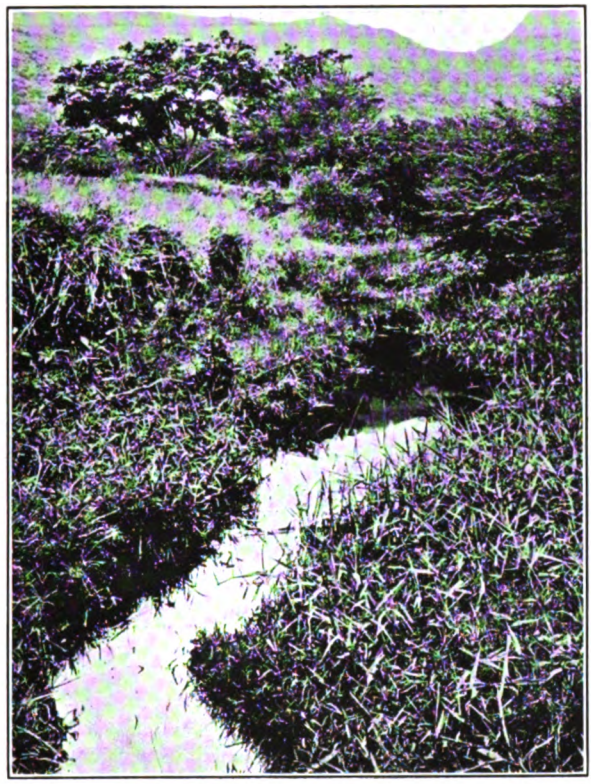

17.—La Paix stream at the time of Ross' and Fowler's visit, 1908.

18.—La Paix Stream. Very nearly the same spot during the dry season at the present date.

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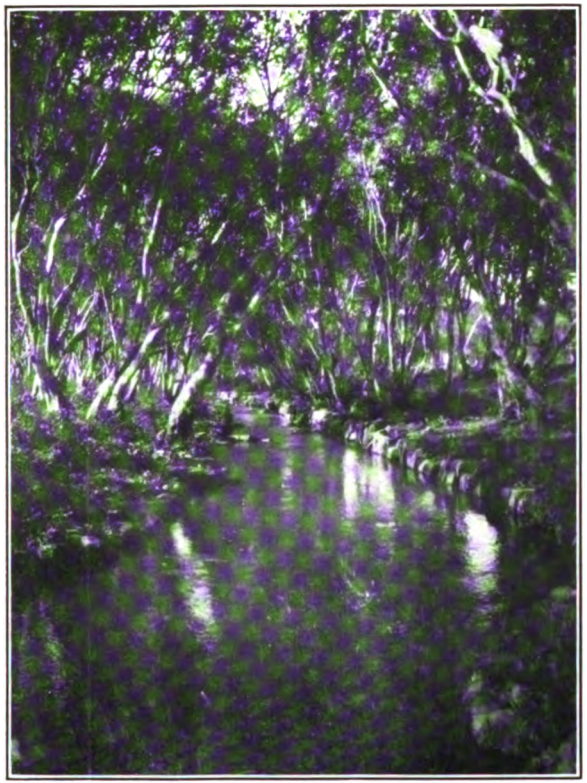
19.—Mosquito trap-pool protected by rabbit wire netting from the assaults of pigs and other animals.



20. Canalised stream shaded by Jamrosa trees (*Eugenia jamrosa*) and free from the larvæ of *A. costalis*.

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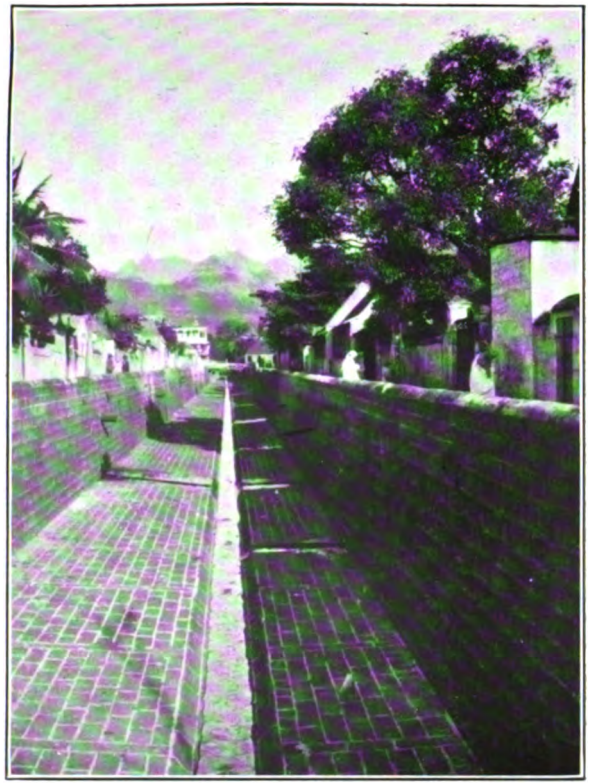
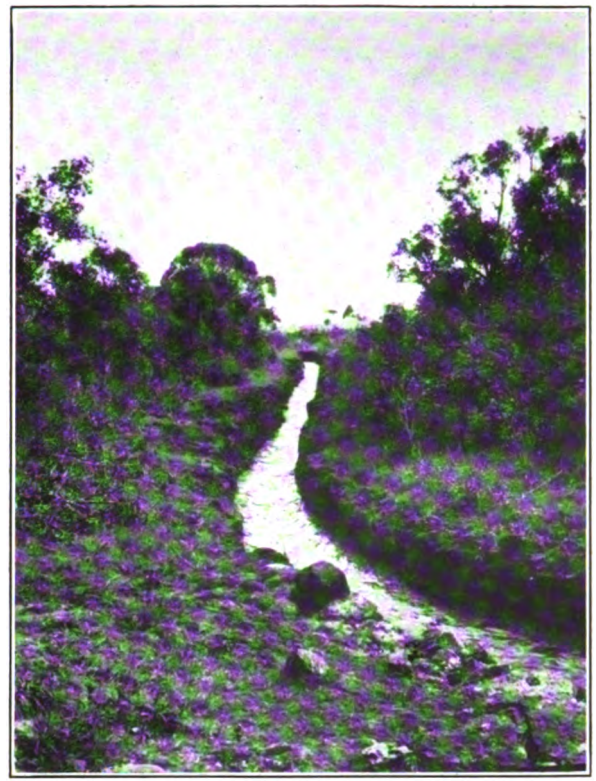


21.—Canalisation of a water-course traversing Port Louis. Note the excellent old Pavé, the side drains and the hill range in the background.



22.—The canalised Rivière Sèche, District of Flacq.

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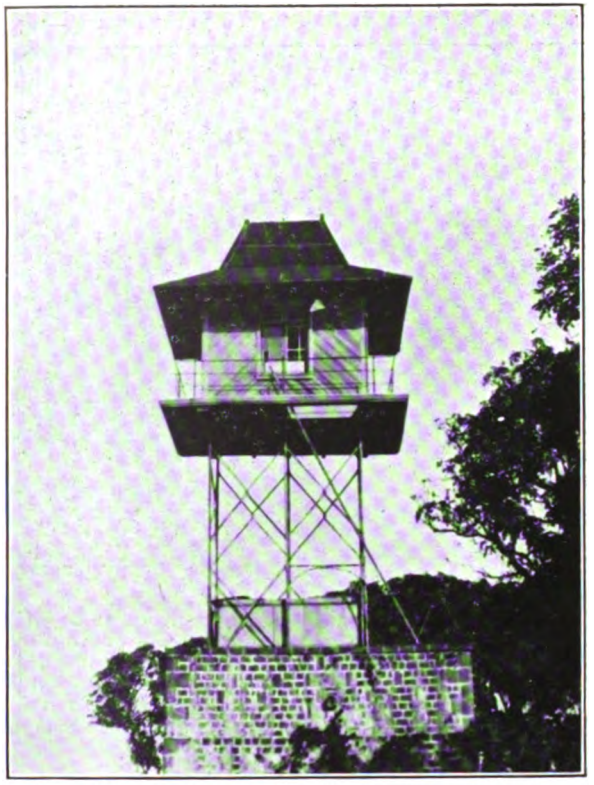
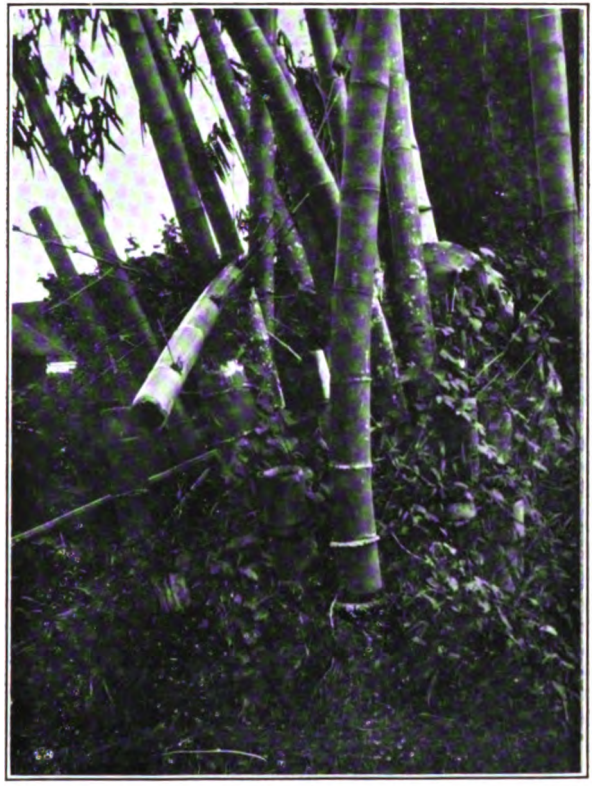


23.—A " mosquito tower " in Pamplemousses district.



24.—Bamboos broken by hurricane and forming nurseries for the larvæ of *Stegomyia scutellaris*.

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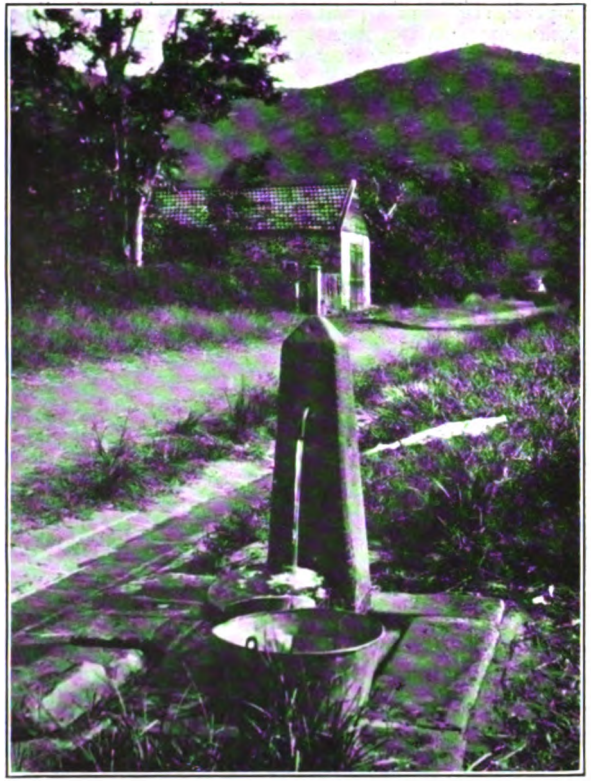
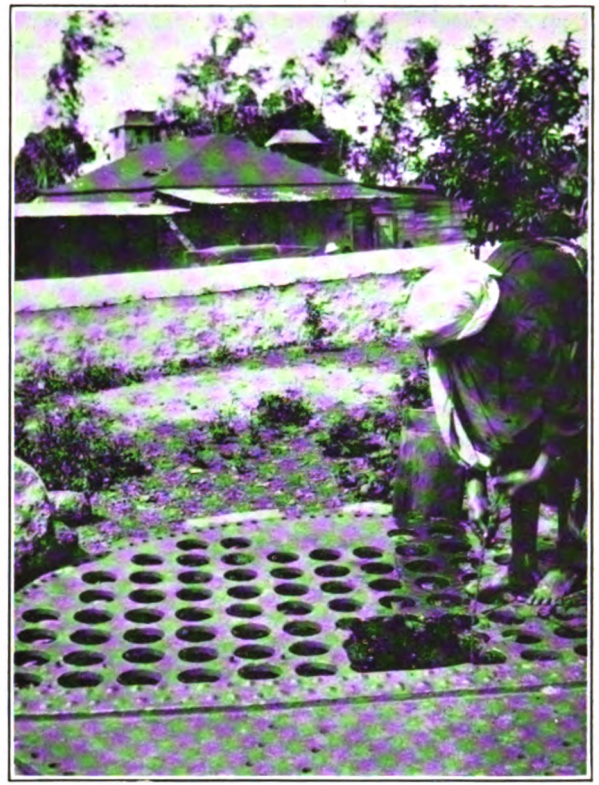


25.—One of the ever-running taps leading not only to a great waste of water, but to the formation of mosquito breeding-places—outskirts of Port Louis.



26.—A well-cover made from the end of a sugar-factory boiler, Ample opportunities for pollution during the drawing of water.

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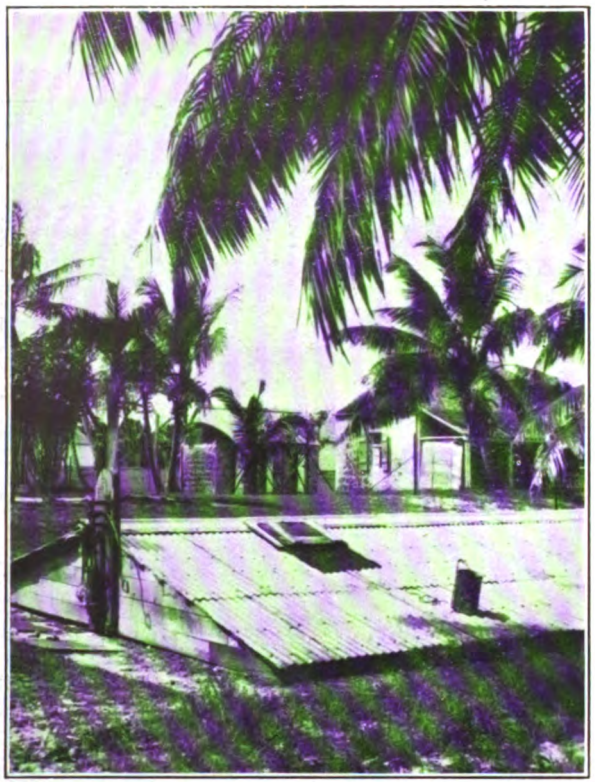
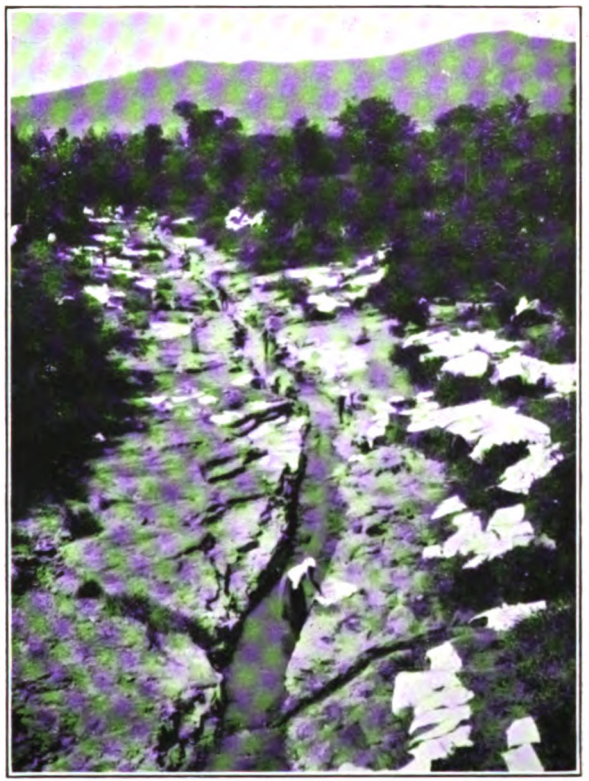


27.—Underground rainwater-tank, Quarantine Station, Canonnier's Point. The water is derived from roof-washings, and as the first washings are not rejected, is somewhat impure. Moreover, the pump had broken down, and consequently resort has been had to dipping, which tended towards pollution of the supply.



28.—Latanier stream near Port Louis, showing native washer-women at work. This photograph also shows how the stream has been canalised in solid rock.

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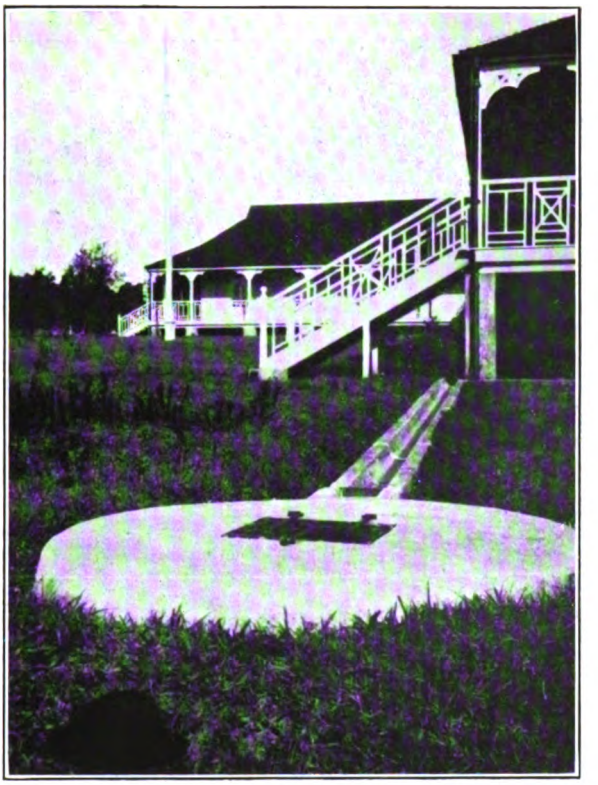

29.—Disposal of waste water. Arrangements at British Barracks at Vacaos. Large covered soak-pit in foreground. The soil here is very porous.

30.—Group of milk-vendors. Tins are of a fairly good type, but their contents only faintly resemble milk, as the product from the cow has had the fat removed, has been boiled, and had water added to it. Nearly the whole milk supply of the island is in a very unsatisfactory condition.

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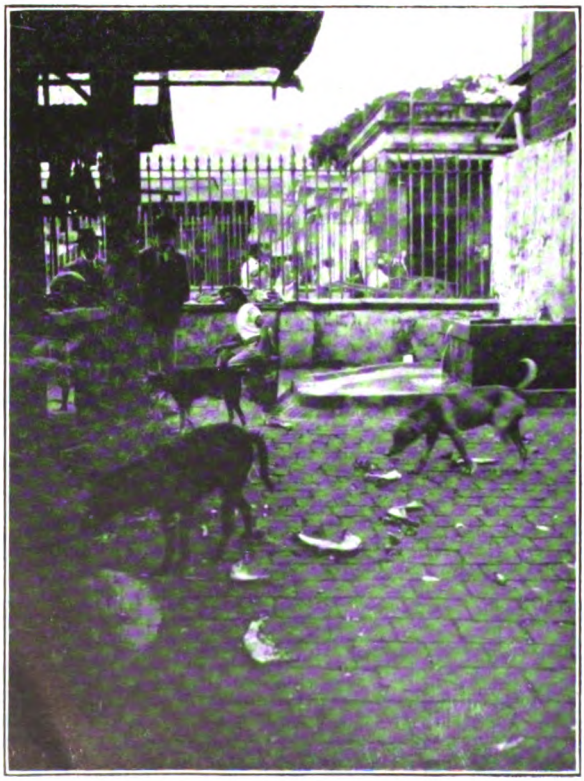
31.—A strange and antiquated vehicle employed for conveying meat from the slaughter-house to the market, Port Louis. It is lined with zinc, but, as will be seen, the fly-proofing is distinctly defective. As a matter of fact the gratings are more for ventilation than for the exclusion of flies.



32.—A corner of the central meat market, Port Louis. A story not needing words

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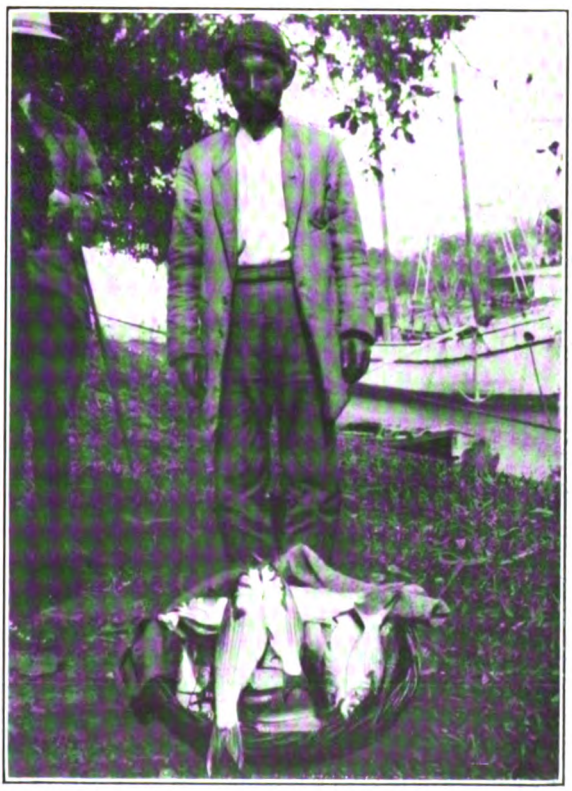
33.—Large covered market in Curepipe. This is the only really good market in Mauritius.



34.—Fish form a staple article of diet in Mauritius, and the photograph shows a basket of fine white mullet.

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35.—A typical night-soil cart, Port Louis. It is furnished with sliding-doors, which render the interior dark. The tubs are of wood, and become very foul.

36.—An open type of engrais or manure pit. The sweeper is shown emptying the contents of a latrine bucket into the cement concrete tank. The human excrement is mixed with cane trash and rubbish of various kinds, and the mass is eventually used for manuring the cane-fields.

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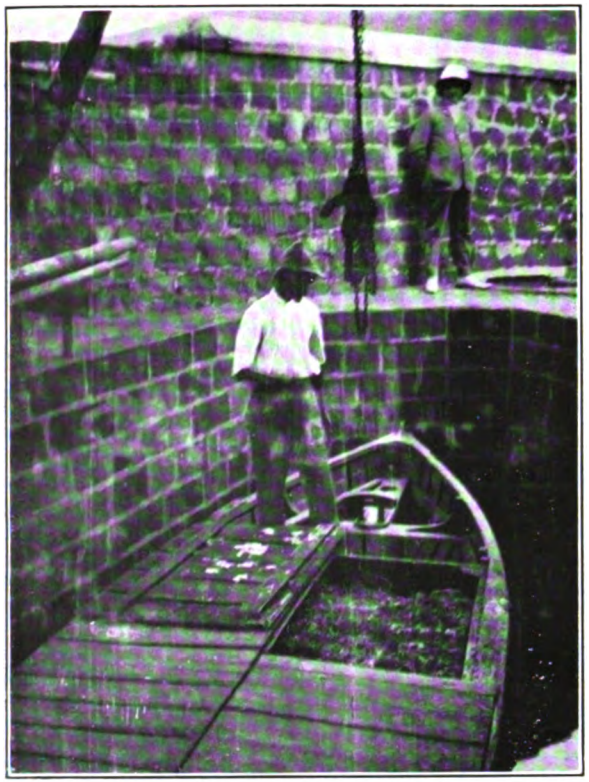
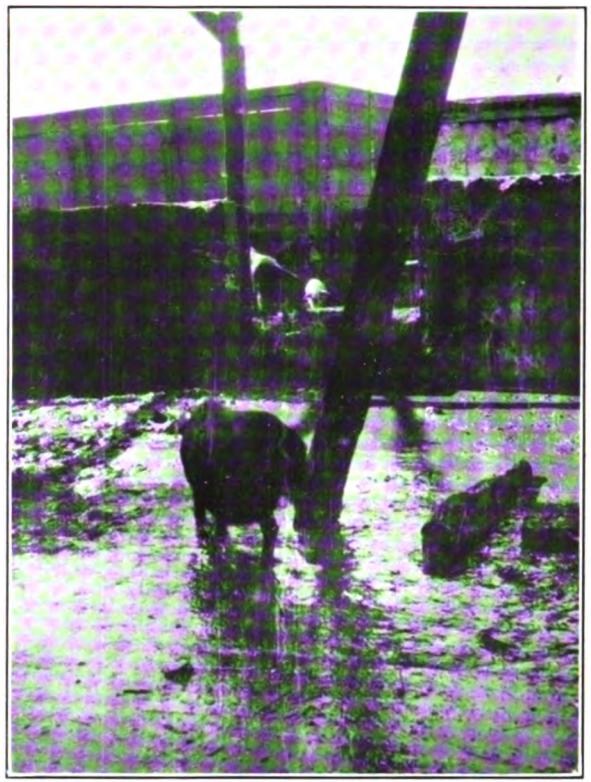


37.—Boat employed for removing a portion of the contents of the sewers, which, owing to partial failure of the pumps and the small size of the outfall are insufficient to deal with the total daily flow. The boat discharges its contents in or outside the harbour and is a happy hunting ground for flies.



38.—Interior of one of the pits at the Roche Bois Manure Factory, Fort Louis. The containing wall is broken and pigs have access to the human excrement.

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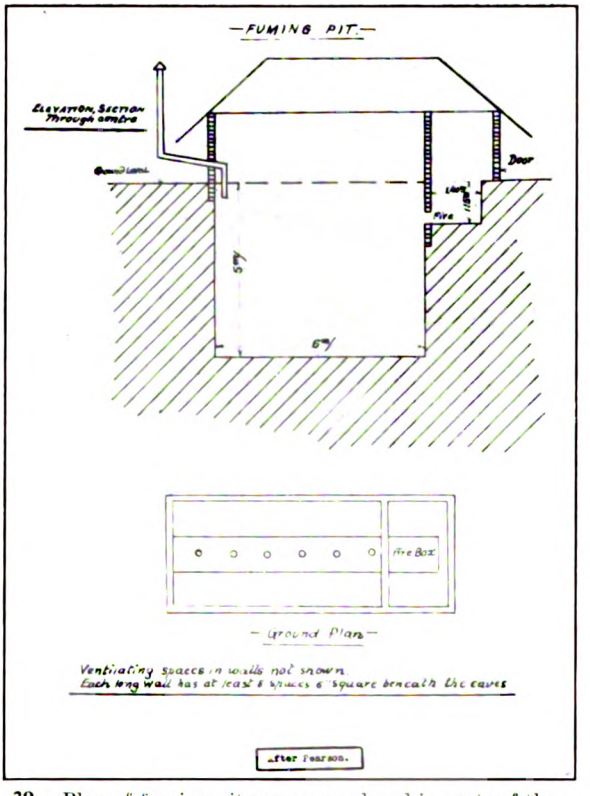

39.—Plan of fuming-pit as now employed in parts of the Belgian Congo. These pits are being introduced into the sugar estates in Mauritius.

40.—A refuse heap and rat playground on the harbour front, Port Louis.

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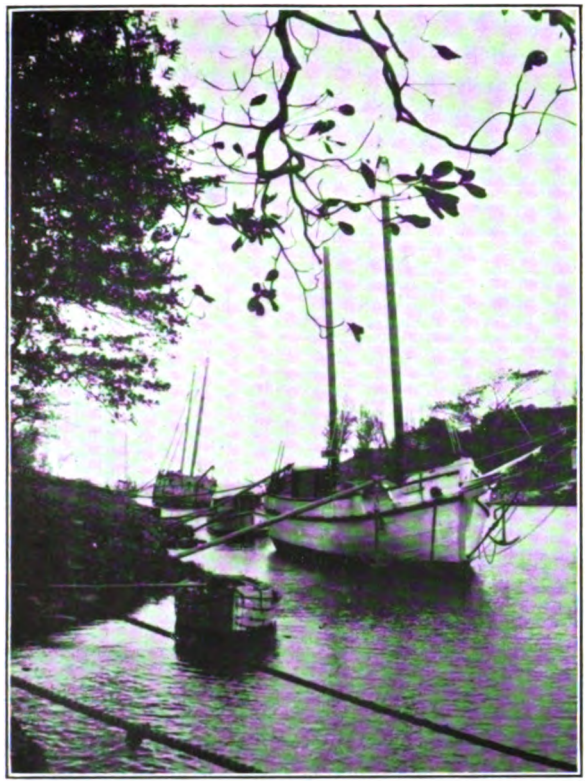
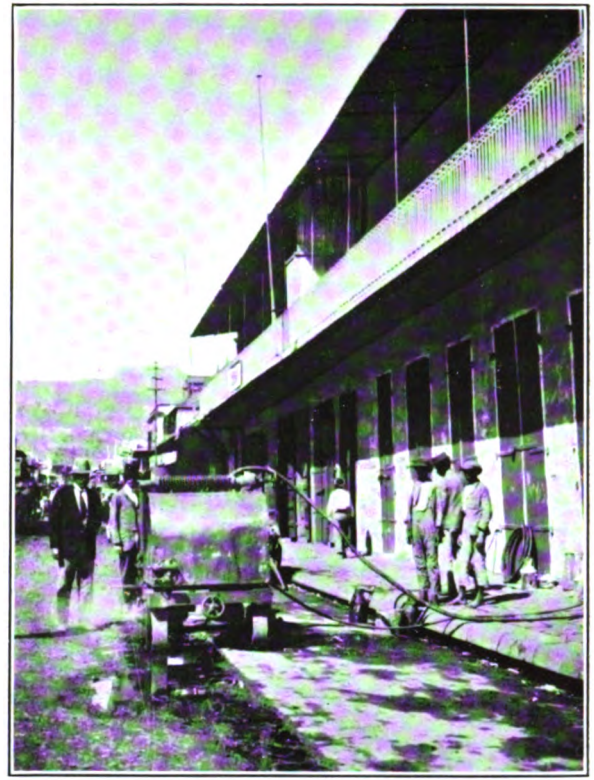


41.—Coasting vessels at Souillac, showing lack of precautions as regards the ingress and egress of rats.



42.—A Clayton apparatus at work during the plague epidemic in Port Louis.

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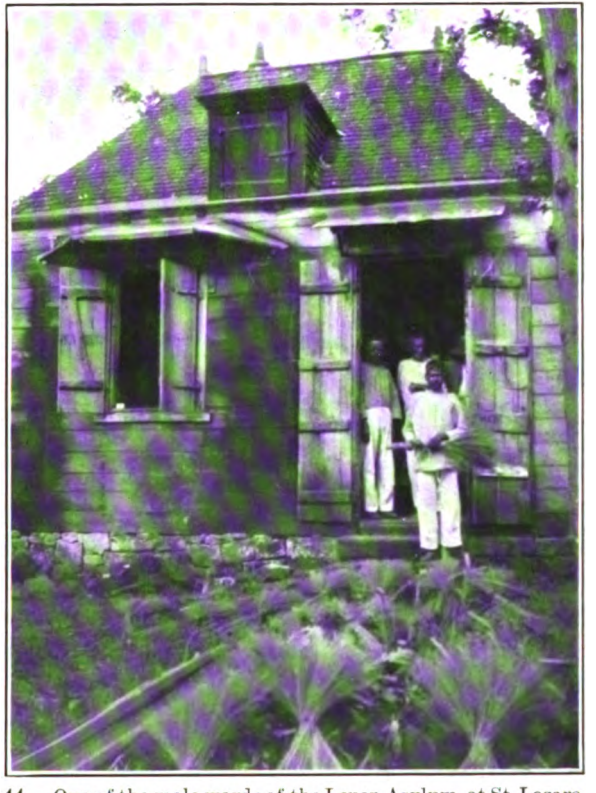
43.—This illustrates the stunting effect of ankylostomiasis on growth. The boy in white is a victim of hookworm disease and is sixteen years of age. The other boy is twelve and was found uninfected.



44.—One of the male wards of the Leper Asylum, at St. Lazare, Port Louis. Note the grass brooms which are manufactured by the lepers, and sold by them to the general public.

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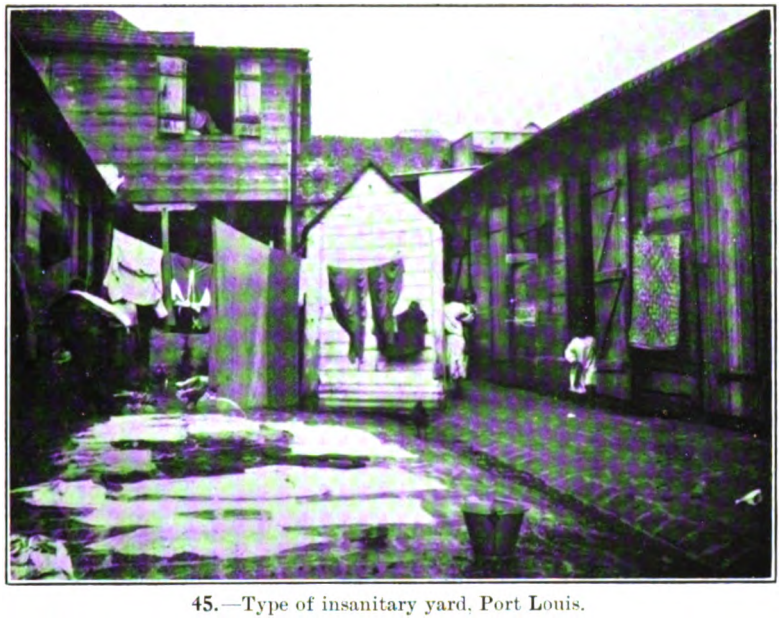

45.—Type of insanitary yard, Port Louis.

46.—Type of better-class Indian's house.

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47.—A Sugar Estate Crèche, where the children are looked after while their mothers are at work.

48.—Portion of the model village being erected by Sir Hesketh Bell at Cassis, on the outskirts of Port Louis.

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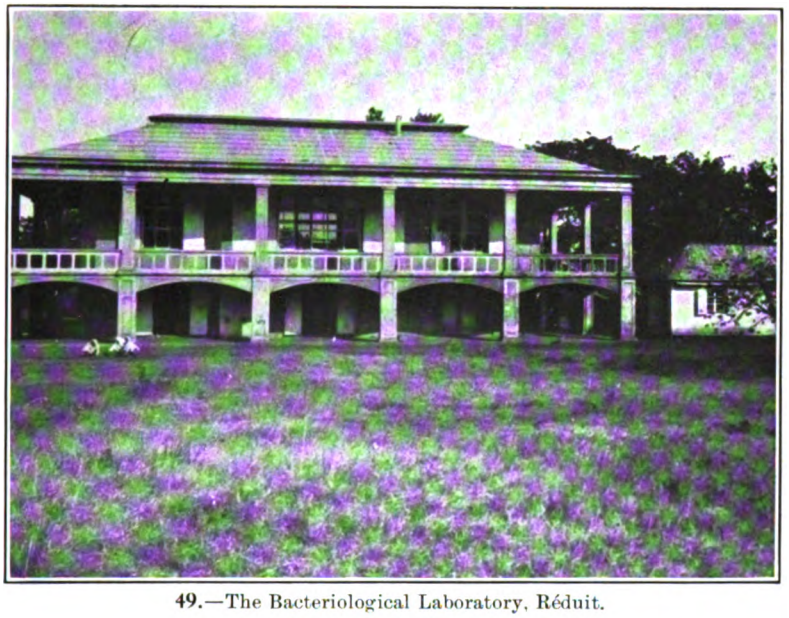
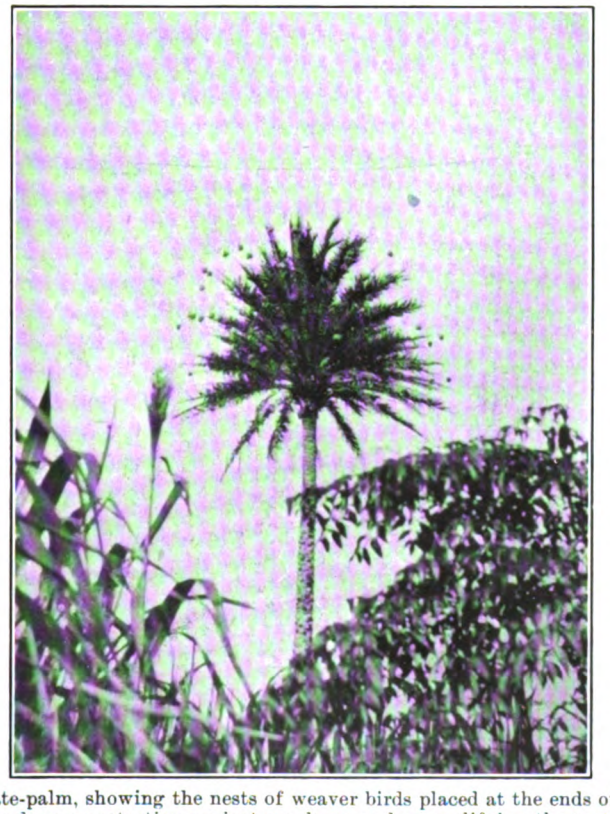


49.—The Bacteriological Laboratory, Réduit.



50.—Date-palm, showing the nests of weaver birds placed at the ends of the leaf fronds as a protection against monkeys, and exemplifying the truth of the proverb, "Prevention is better than cure."

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Malaria.—In the first place, a picture was exhibited of a tombstone in the Bois Marchand cemetery, erected to a large number of the members of the police force who died during the terrible malaria epidemic of 1866 to 1868.

Following this, a series of views was displayed, showing the various breeding-places of *Anopheles costalis*, both in urban and rural districts. So far as the former is concerned, the liability of the so-called "Regards," which are prevalent in Port Louis, to form mosquito nurseries was made the subject of comment. It was explained that a "Regard" was a pit in the street, containing the junction of the pipe from the water-main and the supply-pipes to the house. Owing to the fact that the water of Port Louis, being unfiltered, contains much silt, a blockage is apt to take place at the point of junction. The householder endeavours to remedy this by tampering with the screw-down taps, and, as a result, leakage frequently occurs, and mosquitoes gain access to the water in the pit. Mention was made of a device designed to take the place of the "Regards" and render them unnecessary.

The rural breeding-places comprised swamps (both marine and inland), rivers, streams, ponds, irrigation pits and canals, and special attention was directed to the fact that *Anopheles costalis* does not breed in streams shaded by trees, because the green alga on which its larvæ feed is not found in such situations. Mention was made of Dr. CASTEL's observations in this connection.

A map was then exhibited which showed the very extensive major antimalarial works which had been carried out in the island since Major Ross's visit, and a further idea of what had been done was given by a number of slides showing various kinds of antimalarial operations, more especially those connected with the training and canalisation of rivers and streams. In two instances it was possible to compare pictorially the conditions at the time of Ross's visit and those which now obtain, and a very marked change was visible in the case of a stream in the neighbourhood of Port Louis and a river in the Black River district. The question of *Lemna minor* as a mosquito deterrent was discussed, and the statement made that the work of the SERGENTS in Algiers had apparently shown that this water-weed has not the beneficial effect of *Azolla filiculoides* in preventing ovipositing in sluggish streams and ponds.

A picture of a trap-pool was shown, and the use and value of these

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contrivances explained; while a mosquito-tower was displayed somewhat similar to those used for preventive purposes at certain railway stations in the Caucasus. Other antimalarial measures were discussed, and attention was then directed to—

Non-malaria-carrying mosquitoes and the measures taken to deal with them.—The rôle of *Culex fatigans* as a carrier of filariasis was mentioned, and the statement made that in all probability *Stegomyia albopicta (scutellaris)*, which is so common in the island, was also a vector, as was known to be the case in Malaya. One of its favourite breeding-places, namely, flower-vases in cemeteries, was shown, in addition to such nurseries as snail-shells, barrels, and broken bamboo stems.

Water Supplies—Pictures showing the aerating and oxidation plants at the La Marie Filter Works paved the way for the consideration of many matters connected with water supply in Mauritius. The source, storage, and distribution of water were all passed in review, and pictures illustrating fouling of well-waters formed a feature of this section Comment was made on the waste of drinking-water and its attendant evils, and the measures taken to remedy these evils were explained and illustrated.

Washing.—The manner in which streams and rivers are fouled by the custom of washing clothes in them was next shown on the screen, together with a picture of an open-air laundry.

Waste-water Disposal.—Under "Waste-water Disposal" a single slide was exhibited, which showed the excellent arrangements in connection with the British barracks at Vacoas, where the great porosity of the soil enables well-constructed absorption pits to function admirably.

Food.—Examples of different kinds of food-stuff were shown on the screen, both the necessities and luxuries being represented. Special attention was directed to the question of milk supply, and pictures of cowsheds and milk sellers helped in the elucidation of the subject. The lecturer ranged from slaughter-houses to markets, and the insanitary condition of the central market in Port Louis, and the means taken to remedy it, figured prominently. The excellent, large, covered market at Curepipe received a meed of praise, and then attention was directed to various—

Conservancy Problems, and pictures were shown illustrating the comparative failure of the sewerage scheme in Port Louis, its success in connec-

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tion with the British barracks at Vacaos, and the deplorable conditions existing in connection with the pail system of removal, so general in the island, and more especially the disgraceful state of matters at the Roche Bois Manure Factory near Port Louis, and the somewhat similar establishment which serves Curepipe. The so-called Engrais system was explained and condemned, and some of the remedial measures proposed were illustrated by appropriate slides, notably, the introduction of the "fuming pit" or smoke latrine system, as now practised on a large scale in the Belgian Congo.

Refuse and Refuse Disposal.—These received attention, and the danger of faulty scavenging in connection with the spread of plague by rats was indicated by several illustrations. This led to the exhibition of a series of slides connected with—

Plague and the Anti-Rat Campaign in Port Louis. Amongst others, the Clayton apparatus was shown at work, and the possibility of danger from island schooners tying up to wharves explained.

Then followed No. 12, a picture showing the stunting effect on growth by—

Ankylostomiasis, which is so prevalent in Mauritius, and mention was made of the aid which is to be rendered by the Rockefeller Foundation. The question of—

Housing was thereafter taken up, and a number of photographs passed in review, illustrating not only various dwellings but showing special institutions, as, for example, a crèche on a sugar estate, different kinds of hospital wards, and the model village recently erected by the Governor, Sir HESKETH BELL, at Cassis, near Port Louis.

Pictures of dipping tanks for cattle were also exhibited, and special attention was drawn to a photograph of the useful Bacteriological Laboratory at Réduit, of which Dr. BARBEAU is in charge. Its value from the point of view of preventive work was emphasised, and, finally, the lesson that prevention is better than cure was inculcated by a picture of a date-palm, which showed at the very extremity of its fronds the nests of weaver birds placed there as a protection against the attacks of monkeys.

In the course of the demonstration tributes were paid to the work of several Mauritian doctors and entomologists. The lecturer acknowledged the assistance rendered him by many officials, notably his Excellency the Governor and Mr. E. B. DENHAM, C.M.G., the able and energetic Colonial

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Secretary. Mention was also made of the good work which was being accomplished in Port Louis by the recently appointed Medical Officer of Health, Dr. T. B. GILCHRIST, who would soon be a Fellow of the Society, and of the advantages likely to accrue from the recent visit of a Consulting Sanitary Engineer in the person of Mr. E. L. MANSERGH.

If only Mauritius were freed from those plagues we have considered, and others which render it notoriously unhealthy, its future might, I believe, be assured. There could be no more delightful holiday resort, and no great distance away lies the Union of South Africa, a large and wealthy country developing by leaps and bounds. It has a population, especially in Johannesburg, which looks for rest and change, and nowhere could these be better enjoyed than in the old home of the dodo. At present people are deterred from going there owing to its unenviable reputation for unhealthiness, but if this were abolished there is no reason why hotels should not be built, large golf links laid out and various developments undertaken.

It is possible that thereby the island might lose some of its charm, but it would benefit financially, and it might be expected that its hygienic condition would materially improve, if only because further funds would be available for the upkeep of sanitary works.

All this is no vain dream. We know what has been done elsewhere, wherever modern knowledge has been applied vigorously and along sound lines.

It is only by hard and incessant work that Mauritius can be rendered healthy, but its transformation from a disease-stricken colony to one noted for salubrity is undoubtedly a feasible proposition. It is a question of money, energy, co-operation, education, application of scientific principles, and persistence. The thing can be done, and it is certainly worth doing.

Hence let us hope that now a start has been made under more favourable auspices than in the past there will be no back-sliding, but a steady progress towards the twin goals of health and efficiency.

In the olden days Mauritius, the ancient Isle de France, gained for itself the proud title of the Star and Key of the Indian Ocean. I hope yet to see it become, as apparently it was many years ago, a veritable Sanatorium, a Star of Hope to the sufferer, a Key to the Door of Health.

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Discussion.

The Chairman: I think we must congratulate Dr. Balfour on that very fine lecture which he has just given us, and the beautiful photographs. You will excuse me for saying that it has delighted me beyond measure to hear all these things, and to see in these pictures parts of Mauritius which I had never seen before when I spent nearly fourteen months there with Major Fowler. At that time we had only to deal with malaria. I have not heard much of what happened in the island since then. Mr. De Charmoy was there at that time, and I believe he is still there, though not at the same work. They have not carried out all the recommendations in my report. Dr. Castel, the head of the malaria part of the work, ought to have had his attention confined to malaria. They take one's advice in part, but not fully.

I am glad to hear the island has progressed, and that there has been some improvement, though, apparently, not such great improvement as has occurred in Cyprus. I went to Cyprus in 1913. and there the reduction of disease had been very marked.

I am delighted to hear Dr. Balfour mention my proposal that we should introduce *Anopheles rossi* into the island; but they would not allow it. I do not know why. I only thought of it just before leaving; I had not time to press it. But I think it might eat out the *costalis*. As to the Mauritianus, I do not think that carries; we could not get a single malaria parasite to develop in that, but the first *costalis* we caught was infected.

All the other sanitary details which Dr. Balfour has given us are exceedingly interesting, and he has shown his usual wit and dash in his lecture, and, as I have said, we must congratulate him on it.

I shall not say any more, but I will call upon Dr. Laurent, who was formerly mayor of Port Louis; he was mayor of that town when we were there, and he helped us very largely. I have a great opinion of the municipality of Port Louis. They have an Opera there, which shows that they look after their sanitation, and Dr. Laurent was looking after the sanitation very considerably when I was in the island.

Dr. Eugéne Laurent: I am pleased to meet here an old friend, Sir Ronald Ross, and to make a new acquaintance, Dr. Andrew Balfour; because during my stay in Mauritius Dr. Ross was sent as an

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expert, and he did very good work there indeed. He is right when he says the Government never helped him a bit, and he knows that practically all the work which has been done has been done by the municipality. In the drainage, for instance, we had a splendid expert, Sir OSBORN CHADWICK, who certainly gave the necessary directions, and even left Mr. BRINE to carry out his directions, yet the whole thing was messed considerably by the Government, who tried all the time for economy, and especially delays. The Government has been at the drainage of the island for thirty-one years, and Dr. BALFOUR has given you instances of what the Government did in Mauritius for sanitation. Beyond this, you know that in the suburbs of Port Louis, which might well be called the ante-room of death, the Government has full sway. You will remember, Sir, that I took you round the streams of Rushbury, Maure Rouge, Valley Piteau, and so on, and you saw that all those places had been sadly neglected; so much so that the municipal zone was overcrowded by people who had migrated from the Government zone. I think that is a crucial test. If people migrate to the maligned municipal zone to escape malaria, that is the answer to those who criticise the municipality. Had there been sufficient money available, I think the excellent directions given by Sir RONALD ROSS would have been more successfully and thoroughly carried out by the municipality. I wanted, in 1917, before I left the island, to have a clear indication of the work done by the Government and by the municipality; so I got our sanitary wardens to take the spleen rates in the different schools in the municipal zone and in the zone directly under the control of the Government. The schools under our control gave a 10 per cent. spleen index, whereas on the Government side it ran up to 74 per cent. So that evidently the work done since 1908, the date of the visit of Sir RONALD ROSS, was much more efficient when done by the municipality than was done by the Government. Dr. BALFOUR has forgotten to tell you that before 1867 the island was certainly a sanatorium. But we have now had introduced there so many Indians, for working on our sugar plantations, that apparently all the diseases upon earth were introduced at the same time to our native coolies. And, somehow, the Medical and Health Department, under the control of the Government, did nothing to prevent the introduction of disease, and when disease was introduced there was the greatest difficulty in eradicating it. I think that with the new

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instructions given by Dr. BALFOUR, and with the co-operation of the public, something useful will be done. In fact the municipality, now having more money, will be able to do much of what was left undone owing to lack of sufficient means. You know that in 1908 we had to get Commissioners in Mauritius to look into our finance; the Government had been so improvident that we were on the verge of bankruptcy. But, even at that time, the municipality had no deficit, whereas the Government had more than a million pounds deficit yearly. So, altogether, you see the management was not so unwise on the part of the free institution.

I am sure you will join me in congratulating Dr. BALFOUR on a most useful report, which he has presented this evening, on Mauritius. Everything was very interesting indeed, and, for my part, I enjoyed it thoroughly. I beg to propose a vote of thanks to Dr. BALFOUR, who was your worthy successor.

This was carried, and Dr. BALFOUR very briefly acknowledged it.

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SHORT PAPERS.*

A PRELIMINARY NOTE ON BILHARZIOSIS INDIGENOUS IN PORTUGAL.

By CARLOS FRANÇA M.D., *Naturalist to the Bocage Museum, Lisbon.*

An indigenous case of Bilharziosis, due to *Schistosoma hæmatobium*, was recently discovered at Tavira (Algarve, Southern Portugal) by the urologist, Dr. Bastos Lopes. Several other similar cases were subsequently diagnosed and studied in the same locality by Dr. Machado d'Almeida. As a result of these discoveries, I undertook to study the matter from the parasitological standpoint.

I have found that one of the snails occurring in this district—*Planorbis corneus* L. var. *metidjensis* Forbes, an Ibero-Algerian variety—exercises a very definite chemiotactic action upon the miracidia obtained from the eggs passed in the urine of some of these indigenous cases; and I have further been able to observe the penetration of the miracidia into the bodies of these molluscs. Contrary to Lutz's observations on *S. mansoni*, I find that the miracidia of *S. hæmatobium* do not select the tentacles as their point of entry, but usually enter the snout, the margin of the foot, or even the gill.

I have observed that the miracidia, which are so strongly attracted by *P. corneus metidjensis* (a species containing abundant hæmoglobin), are attracted only exceptionally (1 out of 60) by *P. albus*. *P. crista*, *Physa acuta*, and *Limnæa peregra* are not attacked at all. In one specimen of *P. albus*, obtained from a locality where there is no bilharziosis, I have found bifid-tailed cercariæ without eye-spots; but I have not yet been able to identify them. In the pond from which our cases appear to have acquired their infection I have as yet succeeded in finding only two species of snail—*Physa acuta* and *P. corneus*. Of the latter species, only dead specimens are, as a rule, discoverable.

*With a view to making the Transactions more interesting, it is proposed to insert SHORT COMMUNICATIONS from Fellows relative to any matters of medical, sanitary or parasitological interest. It is felt that many Fellows in various parts of the world must be in a position to contribute short papers on these subjects, and the Secretaries will be pleased to receive any such from them.

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So far as I have been able to ascertain up to the present, the hatching of the eggs of *S. hæmatobium* takes place with great difficulty in waters from granitic soils. My experiments indicate that the great majority of individuals of *P. corneus* die within a short time after being attacked, and in such dead specimens I have found well-developed sporocysts. This pronounced pathogenic action of *S. hæmatobium* upon *P. corneus* doubtless explains the slow spread of bilharziosis in our country.

Frequency curves based upon the lengths of the eggs of *S. hæmatobium* show certain points of interest, and I am pursuing this line of investigation further. The eggs usually measure 133μ to 143μ in length; but their dimensions are variable, and the variation seems to follow a definite law, which is shown in the frequency curves that I have constructed. These curves appear to have some importance in connection with the interpretation of the effects of treatment.

Although we are still only at the beginning of our investigations, we believe that the results which we have so far obtained are worthy of immediate publication. Winter has already commenced, and it will therefore be impossible to finish our study of the problem this year. Our results appear to us, moreover, to raise a question of very great hygienic importance—that of the possible acclimatization of bilharziosis in Europe. Fears were formerly entertained that the Schistosomes might spread to Europe; but these fears seem to have been set at rest by the researches of LORTET (1905), and especially by those of ROUBAUD (1918). Our observations appear to us to open up the whole problem anew.

Fortunately, *S. hæmatobium* appears to be established at present in only a very small area in Portugal; and it seems legitimate to hope that, before long, the disease will become extinct among us. The lesson will, however, remain; and it clearly teaches us that imported carriers of bilharzia may constitute a source of danger to Europe unless they are rendered innocuous by treatment with tartar emetic.

In conclusion, I desire to thank Mr. CLIFFORD DOBELL for his kindness in helping me to prepare this note for publication in English.

COLLARES, PORTUGAL.

8 October, 1921.

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ON SOME HELMINTH PARASITES OF AN EAST AFRICAN ROCK RABBIT (PROCAVIA SP.).

By Miss M. TURNER, M.Sc.,
Helminthological Department, London School of Tropical Medicine.

During his stay in German East Africa, Captain G. Norman Hall, M.R.C.V.S., of the Veterinary Department, Dar-es-Salaam, made an extensive collection of the helminth parasites of various animals. Through the kindness of Captain Hall and of Dr. Bartlett, of the London Hospital, this collection was forwarded for diagnosis to the Helminthological Department of the London School of Tropical Medicine. Among the specimens were some parasites from Rock Rabbits which had been shot at Dodoma, near Mpapua.

Further examination showed that these included the following nematodes:—*Deletrocephalus brachylaimus*, von Linstow, 1901, *Hoplodontophorus flagellum*, Hemprich & Ehrenberg, 1828, nov. gen., *Crossophorus collaris*, H. & E., 1828.

DELETROCEPHALUS BRACHYLAIMUS, VON LINSTOW, 1901.

Owing to the presence of a very long bursa in the male of this species the two sexes are almost equal in length, but the female is stouter than the male. The anterior end of the body is markedly tapering and the head is small in proportion to the rest of the body.

The cuticle is transversely striated at intervals of from 25μ to 30μ (von Linstow gives 21μ).

The œsophagus is from 300μ to 375μ in length. It has a constriction at from 80μ to 125μ from its anterior end and where the nerve ring is situated; behind this the œsophagus dilates until it reaches a diameter of from 125μ to 200μ.

The position of the cervical papillæ is characteristic for they are from 800μ to 840μ from the anterior end of the body; this means that they

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are situated almost one and a half times the length of the œsophagus behind the commencement of the intestine. The excretory pore opens in front of the cervical papillæ at about 650μ from the anterior end of the body.

The wall of the intestine, except at its commencement, is impregnated with a brownish pigment.

A mouth collar is present and is relatively high, being from 22μ to 30μ in height. At its base it is 104μ to 130μ wide, while at its anterior end it is from 80μ to 105μ wide: in optical section its lateral margins are trilobed.

The lateral head papillæ end anteriorly in the middle lobe of the mouth collar and do not project far. The submedian head papillæ project in front of the mouth collar, and each consists of a conical base, from which arises a narrow club-like process.

The mouth capsule is ellipsoidal in shape, with its longer axis dorso-ventral. In optical section the mouth capsule walls are from 15μ to 25μ in height, and vary from semi-circular to triangular in shape. The breadth of the mouth capsule is from 60μ to 65μ laterally and from 75μ to 80μ dorso-ventrally.

On the anterior end of the œsophagus is a rim of cuticle into which the mouth capsule fits. In optical section this rim appears as a small pointed projection on each side of the mouth capsule.

Arising from the level of the base of the mouth capsule is a corona of about thirty pointed leaves. Owing to its ellipsoidal shape, this corona appears wider in lateral view than in dorsal or ventral.

In his description of *D. brachylaimus*, VON LINSTOW has apparently mistaken the identity of the several parts of the head region: thus he speaks of "four large papillæ at the base of the head prominence, in the submedian lines." As far as can be made out from his figure these "papillæ" seem to be the optical sections of the mouth capsule. Also, he has evidently taken the fibres leading to the submedian papillæ for the optical section of the mouth capsule, and mistakes the leaves of the corona for rods in the wall of this "capsule."

Female.—VON LINSTOW gives the length of the female as 20mm. and its breath as 880μ. In the material from German East Africa, the mature females measured from 11mm. to 15mm. in length and from 700μ to 725μ in breadth. The tail is rounded with a small point at its

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DELETROCEPHALUS BRACHYLAIMUS
(V.L. 1901).

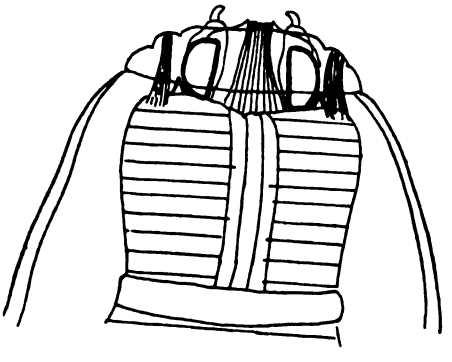


Fig. 1.
ANTERIOR REGION OF BODY.

HOPLODONTOPHORUS FLAGELLUM
(H. & E. 1828).

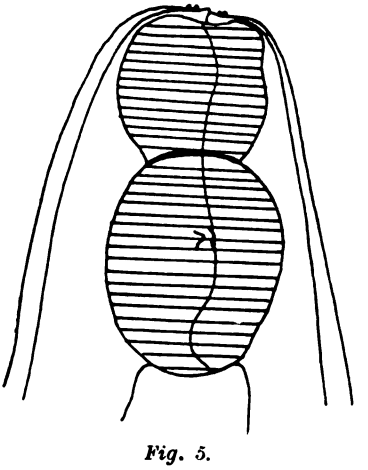
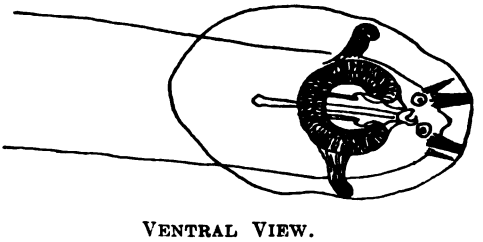


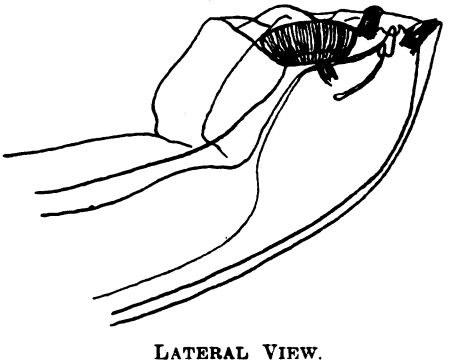
Fig. 5.
ANTERIOR END OF BODY.

HOPLODONTOPHORUS FLAGELLUM (H. & E. 1828).

Fig. 6.
POSTERIOR REGION OF MALE.



VENTRAL VIEW.



LATERAL VIEW.

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DELETROCEPHALUS BRACHYLAIMUS (V.L. 1901).

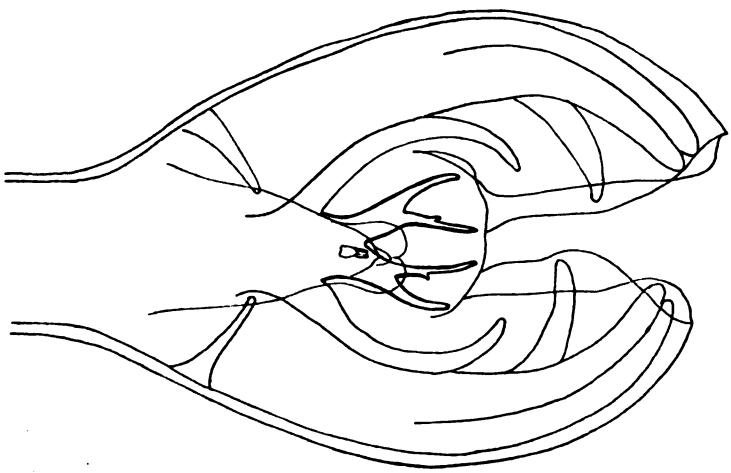


Fig. 2.
DORSAL VIEW OF BURSA.

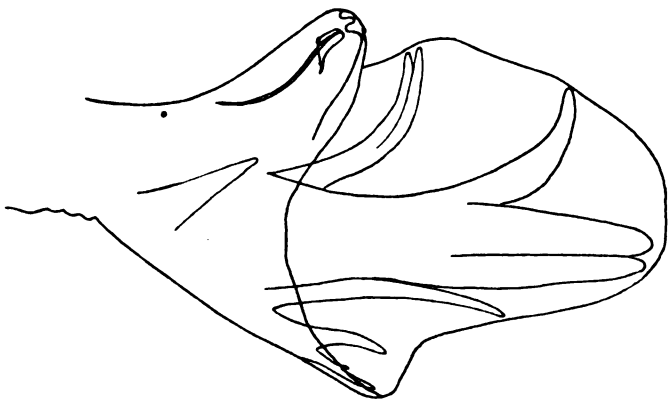
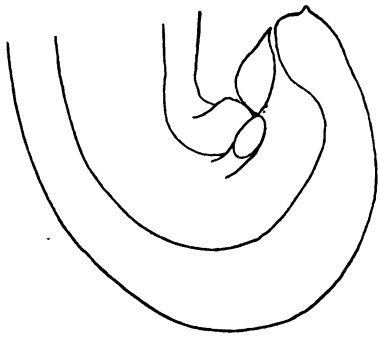


Fig. 3.
LATERAL VIEW OF BURSA.

POSTERIOR REGION OF FEMALE.



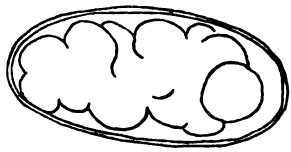


Fig. 4.
EGG × 220.

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extremity; it is variable in appearance, for in some specimens a large ventral prominence is seen just in front of the anus, while in others the tail is bent ventrally. In some females an outstanding cuff of cuticle, about 900μ in length, is present in this region.

The anus is from 75μ to 100μ in front of the terminal point; while the vulva, which sometimes opens at the apex of a prominence, is about 200μ in front of the anus.

The eggs are oval and thin shelled: they measure from 150μ in length by 65μ in breadth to 152μ in length by 72μ in breadth.

Male.—The total length of the male is from 9·5mm. to 13mm. and of this 1·25mm. to 1·55mm. is accounted for by the bursa. The breadth of the body at its widest part is from 450μ to 550μ.

The genital cone is very long, with a well developed dorsal collar. The pre-bursal rays are thick and pointed.

A gubernaculum is present.

The two spicules are thin and are from 3·1mm. to 3·5mm. in length (VON LINSTOW gives 2·96mm); each has a simple point at its distal end.

The bursa has two large lateral lobes, which are from 1·25mm. to 1·55mm. long, and a smaller median lobe which is about 0·9mm in length. The dorsal ray is bifid, each part dividing into two branches, of which the internal bears a small process on its outer side. In my material this internal branch is only slightly longer than the external one, but VON LINSTOW figures it as over twice as long as the latter. VON LINSTOW also states that the externo-dorsal and dorsal rays apparently arise separately, but in my material they arise together, and the externo-dorsal ray curves away from the dorsal just before the latter bifurcates.

VON LINSTOW figures the cleft between the internal lateral and the median lateral rays as about twice as long as that between the latter and the externo-dorsal. In my material these clefts are of equal length. The ventral rays are not separated throughout the proximal half of their length.

HALL says that it is not certain that *D. brachylainus*, VON LINSTOW, 1901, is congeneric with *D. dimidiatus*, DIESING, 1851, which is the type species of the genus Deletrocephalus. The fact that in the latter there are six projections from the base of the mouth capsule, which are

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absent in the former, is an important point of difference between the two forms.

HOPLODONTOPHORUS FLAGELLUM (HEMPRICH AND EHRENBERG, 1828). NOV. GEN.

HEMPRICH and EHRENBERG first described the female of this nematode from *Hyrax syriacus*, and called it *Oxyuris flagellum*. However, on the discovery of the male in my material, it was decided to remove it from the genus Oxyuris and place it in a new genus Hoplodontophorus, on account of the presence of a ventral sucker in the male.

Definition of *Genus Hoplodontophorus*: Male with peculiarly sculptured horseshoe-shaped sucker on the ventral surface anterior to the cloaca. Well developed bursa present. Œsophagus as in *Oxyuris curvula*, short and with an œsophageal bulb. Mouth surrounded by six pointed inconspicuous lips.

Description of *Hoplodontophorus flagellum*. The male is only from one quarter to one half as long as the female, and about half as broad.

The œsophagus is short, and approximately equal in length with the œsophageal bulb. The nerve ring is situated about the middle of the œsophagus.

HEMPRICH and EHRENBERG probably mistook the small points of the lips for papillæ.

Female.—The female is from 17mm. to 32mm. long and from 1mm. to 1·2mm. broad in the region of the vulva. Just behind this the body becomes slightly broader and then gradually tapers to the anus. A short distance behind the anus the body narrows abruptly to form the long, thin tail, the distal end of which is rounded and ends in a very small point.

The œsophagus is 0·3mm. in length by 0·25mm. to 0·3mm. in width. The œsophageal bulb is from 0·35mm. to 0·45mm. long by from 0·3mm. to 0·35mm. wide.

The vulva is from one-fifth to one-third the length of the body, from the anterior end: its lips are swollen and project from the body wall. The ovaries are found in the region of the vulva. Their ducts pass backwards to a point just in front of the anus, where the large uterus commences. This passes forwards to a level anterior of the vulva, where it turns backwards as the short vagina.

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The anus is from one-fifth to one-third of the length of the body from the extremity of the tail.

The eggs are flattened on one side and measure from 110μ by 42μ to 115μ by 45μ.

HEMPRICH and EHRENBERG note two "varieties" of their *Oxyuris flagellum*: (*a*) "obtusa," which is yellow in colour, and has a curved tail that is more than one-third of the length of the body, and (*b*) "acuta," which is white in colour and has a straight tail which does not exceed one-third the body length.

In the material from German East Africa, both white and yellow forms are present. The latter are packed with eggs while the former contain very few. This seems to account for the difference in colour. In my material, as HEMPRICH and EHRENBERG noted in *Oxyuris flagellum*, the tail of a yellow specimen seems to be longer in proportion to the rest of the body than that of a white one. This, however, is not a specific difference.

Male.—Measures from 7·7mm. to 8·4mm. in length and from 0·5mm. to 0·7mm. in breadth.

The œsophagus is 0·18mm. long by 0·15mm. broad and the œsophageal bulb is 0·25mm. long by 0·2mm. broad so that it is just over half as long as that of the female.

The posterior end of the male for a distance of 0·6mm. is surrounded by a large bursa. This is supported by three pairs of papillæ. The most anterior pair is large and is situated from 0·25mm. to 0·27mm. from the posterior extremity. The papillæ of the other two pairs arise close together at the sides of the tail point. One pair of ventral papillæ are present at the sides of the cloaca.

The tail ends in a short blunt point.

The centre of the sucker is about 0·25mm. from the posterior end. This sucker is quite large with a diameter of 200μ, and is incompletely annular in shape. Its walls are striated, and at one level the striæ are broken which gives the sucker an appearance like that of two horseshoes, placed one inside the other. The gap in the walls is situated caudad. In the central gap of the sucker lie two teeth, symmetrically placed at each side with their points directed posteriorly. At the sides of the gap in the sucker wall are two pointed flaps of transparent cuticle which

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reach to the cloaca which is about 50μ caudad of the sucker. The cloaca is two lipped.

The single spicule is about 340μ long and 15μ wide. Its proximal end is knob-like.

A gubernaculum is present. In lateral view it is shaped like a long arrow head.

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NOTES ON THE INCIDENCE OF FILARIAL INFECTION IN THE NEIGHBOURHOOD OF LAMU, BRITISH EAST AFRICA.

By G. DUNDERDALE, M.D., M.R.C.S.

Introductory Remarks.—On proceeding from Nairobi to assume medical charge of the station of Lamu, I was asked by the Principal Medical Officer to investigate, if possible, the subject of filariasis and elephantiasis in the neighbourhood, with special reference to the marked difference in percentage infections reported by previous Medical Officers from two villages only a few miles apart.

In his Annual Report for the year 1911-1912 the Medical Officer in charge gave the following table :—

Cases examined	118
No. showing filarial infection	42
Percentage infected	35·60

These cases were from the following sources :—

Cases from Lamu itself	67
No. infected	27
Percentage infected	40·30
Cases from Siyu	28
No. infected	14
Percentage infected	50·00
Cases from Faza	23
No. infected	1
Percentage infected	4·34

Particular attention was invited to the striking difference in percentage between the two places, Faza and Siyu, which are situated on the same island, and only a few miles apart. Apparently, however, no work had been done on the question of tribal incidence, and I could find no record of the identification of any particular filaria.

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I therefore decided to attempt to work out, as far as possible, the following points:—

1. Percentage of persons infected.
2. Percentage according to locality.
3. Tribal incidence.
4. Causative filaria.
5. Carrier or other predisposing agent.
6. Relation between filariasis and elephantiasis.

This programme proved much too extensive for completion in the time at my disposal, and the work was handicapped by difficulties in securing clinical material (a large amount of the work having to be done after 8 o'clock p.m.), and by the lack of equipment for research at this station.

The locality provides an ample and compact field for study, and further accurate observations would be of great interest and value. But it is very desirable that the observer should have his whole time free for his research for some months at a time.

Elephantiasis and filariasis appear to exist all along the coast of British East Africa, with the exception of the most northerly portion.

From the Tana River (40 miles south of Lamu) up to Kiunga (about 50 miles north of Lamu) these conditions are to be found, but the localities where they are most common are undoubtedly the islands of Lamu and Patta, and certain areas on the Tana River. On the mainland, except for the Tana River area, the diseases under consideration are far less common, and occur in the majority of cases among island natives who have settled on the mainland.

Cases also occur further south, and some of those examined and dealt with in this paper appear to have contracted their infection at Mombasa and other places in British and German East Africa. In an addendum a few cases from Pemba are dealt with at the end of this paper.

Clinical Appearances.—The common obvious form of the disease elephantiasis is that affecting the legs, known locally as "madende"; scrotal elephantiasis also occurs commonly.

I have seen no other forms of this disease, though some cases of lymphangitis closely resemble it, and may be seen affecting the arms. I class these not as elephantoid lesions proper, but as filarial. Of the latter many are to be seen. Filarial fever is frequent, and lymphangitis and hydrocele common. Lymphangiectasis occurs. I have seen no definite cases of chyluria.

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Apart from such manifestations, however, it must be admitted that the majority of persons who harbour filariæ in their systems present no clinical lesions at all, at least for a large period of their lifetime. Though filariasis occurs, as stated, all along the coast of East Africa, the region round Lamu appears to be a real endemic area, and a note on its climate and physical features, etc., will not be out of place.

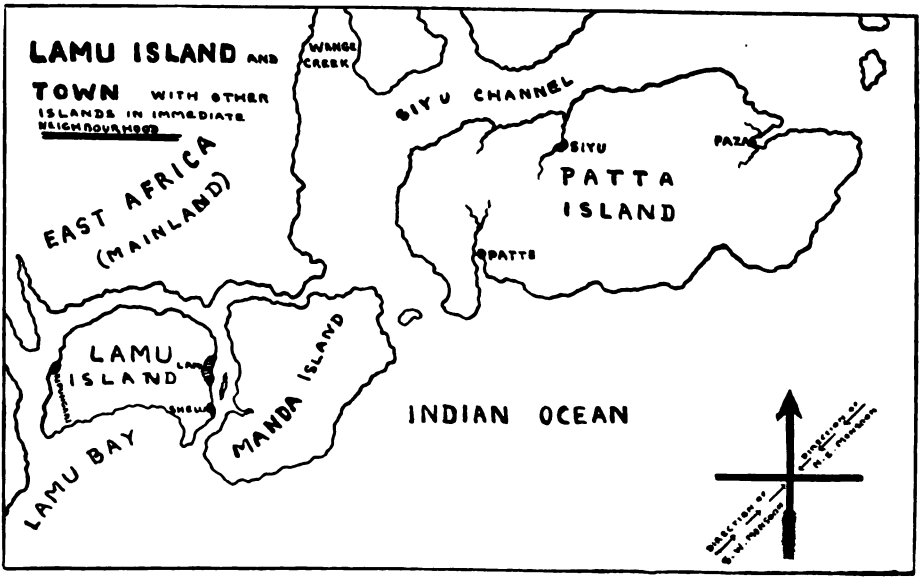
The island of Lamu is situated about two degrees south of the Equator. It is close to the mainland, lying in a small bay, and is separated on its northern and eastern aspects from the Indian Ocean by the island of Manda, which screens a large portion of it from the open sea, and provides it with a sheltered harbour.

The island of Patta lies a few miles to the north-east of Manda, and is rather larger, and fully exposed to the sea on its eastern aspect.

Both islands were much more prosperous in former times than they are now, having been occupied by the Arabs and the Portuguese, and seen considerable trade and activity in the past.

The population is, accordingly, very mixed, and in addition to almost every tribe of East and Central African native there may been found Indians, Egyptians, and Persians, or their descendants. The question of tribal incidence has therefore considerable interest.

A sketch map of the immediate neighbourhood is included to illustrate certain points in this paper.



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Most of this endemic area is flat, with a little rising ground, and some sand hills. The soil is sandy. Nearly the whole of the island of Lamu is cultivated with coconut trees, which are also cultivated largely at Siyu Faza. Mangrove bushes and trees are also plentiful. The climate is healthy and fairly dry. In Lamu, where regular records are taken, the rainfall for the whole year was 34·90 inches in 1914, and 42·51 inches in 1915. It may be considered to average about 37 inches for the year, and is usually rather more on the mainland.

In 1914 the average shade temperatures for the year in Lamu, were: maximum 87·62°, minimum 78·59°, and in 1915 the corresponding figures were 85·08° and 76·56° respectively. The fresh winds prevent any marked sense of heat in the daytime, and the nights are rarely oppressive.

It is stated that two atmospheric conditions, viz., high air temperature and considerable atmospheric humidity, are generally associated with the prevalence of filariasis and elephantiasis. I have no means of measuring the relative humidity in this locality, but the temperature cannot be considered very high for the Tropics. Lamu itself has a sandy foreshore facing east, while Siyu, to which further reference will be made, is situated on a sluggish creek, surrounded by mangrove bushes, and the relative humidity there is probably high. This creek faces west, and is not well ventilated in either monsoon.

The population of the principal towns and villages of the locality is as follows (approximately) :—

Lamu Island	7,000
Faza	2,990
Siyu and Patta	2,000
Mkonumbi (mainland) ...	900
Witu (mainland)	850
Kipini (Tana River)	600

The people are largely occupied in agriculture, or as sailors and fishermen. They show a wide variation, from comparative civilisation to gross ignorance and superstition, and are, on the whole, extremely primitive in their attitude towards medicine and research work. Operative treatment is almost always refused, and post-mortem examinations are hardly ever performed owing to the abhorrence with which they are regarded by the majority of persons of the Mohammedan faith. Operative measures for the improvement or cure of elephantiasis are

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therefore out of the question at present, and it is impossible to avail oneself of the large amount of material for investigating the pathology and morbid anatomy of the lesions grouped under the heading of elephantiasis.

The chief diet of Arabs, Indians, and Africans is rice, but the latter also eat a fair quantity of Indian corn and other cereals. Meat and fish are eaten in Lamu, the latter being very plentiful and cheap,

Elephantiasis is unrecorded among Europeans here, and is uncommon among Indians.

Sanitation in all the areas is bad, and all water supply either contaminated with sewage, or brackish and saline, except in the Tana River district.

Entomology.—Ticks, fleas and lice are not very common in Lamu. Bugs abound in native mattresses and bedsteads.

Mosquitoes.—The following are universal in the district :—*Culex fatigans*, *Stegomyia fasciata* and *Pyretophorus costalis*, and at Siyu there is an exceedingly common mosquito, which has been identified as *Howardina pembænsis*, THEOBALD. *C. fatigans* and *P. costalis* we know are capable of carrying the filarial infection, but I have been unable to obtain personal proof on this point, owing to lack of time and apparatus. Further work remains to be done on the subject.

Causative Filariæ.—Embryonic *Filaria bancrofti* were found in the peripheral blood of all cases returned as infected. Dr. G. C. Low, to whom I submitted specimens, kindly confirmed this for me. All cases were examined after 8 p.m., and many by day as well. In those examined by day, either no filariæ at all were seen, or, at most, one or two to a film. These were exactly the same as those seen in the blood at night. Thick blood-films were taken in the usual way, dehæmoglobinised, and stained with Leishman's stain. This was used not for preference, but because it was always at hand, and I had difficulty in obtaining other stains. Mounted preparations, however, were found to fade very rapidly.

With regard to the relation between filariasis and elephantiasis, I have so far obtained no data of value.

Up to date, 317 cases have been examined in all, and of these 112 show infection with *Filaria bancrofti.* This is a total percentage of 35·33. They may be tabulated as follows :—

Total Cases.	Males infected.	Females infected.	Total infected.		Males free.	Females free.	Total free.
317	97	15	112	...	188	17	205.

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Very few females were examined in proportion to the number of males, and the figures are hardly enough to judge by, but it will be seen that of these few a higher percentage is affected than of the males. Elephantiasis of the legs is very commonly observed in elderly women.

TRIBAL TABLE.

Tribe.	No. examined.	No. infected.	Percentage.
Swahili ...	56	22	51·78
Bajun ...	85	31	36·47
Wa Pokomo	86	20	23·25
Arab ...	43	19	44·18
Kikuyu ...	10	1	10·00
Other tribes... (including 17 different tribes)	37	12	32·43

PERCENTAGE ACCORDING TO LOCALITY.

Place.	No. examined.	No. infected.	Percentage.
Tana River ...	89	20	22·84
Lamu ...	66	32	48·48
Siyu ...	82	36	43·90
Faza ...	27	10	37·04
Other places...	53	14	26·41
(Pemba ... (*Vide* Addendum.)	50	20	40·00)

It is, therefore, evident that in this locality the Swahilis, Bajuns and Arabs are the most generally infected with filariasis, and that Lamu and Siyu are the most heavily infected places. Of other tribes, however, with the exception of the Wapokomo, so few have been examined in comparison that the figures with regard to them are of no value. The proper Coast people, *i.e.*, the three tribes mentioned above, should naturally show a heavier percentage of infection than up-country tribes, who are not permanently resident in an endemic area.

The difference in infection at Faza and Siyu respectively is small in my cases, and contrasts markedly with the figures in the table given in the report for 1911, when the percentage infection at Faza was found to be 4·34. It is hardly possible that so great an increase can have taken place in such a short time. In any case more people must be examined from Faza and Siyu. Natural conditions favour a heavy

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infection at the latter place. If the map be consulted it will be seen that it faces almost due north-west, and is on the western aspect of the island of Patta. It is low-lying, insanitary, and full of mosquitoes. In neither monsoon does it receive nearly as much direct wind as Lamu, Faza, or Patta, all of which face more or less east, and the people are mostly cultivators, who do not travel about as much as the inhabitants of the busier trading places. Accordingly I attribute the prevalence of the diseases at Siyu to physical and local conditions as much as anything. Lamu Island, which actually shows the heaviest infection of all, has a more mixed and moving population. If all inhabitants—Europeans, Indians and natives—were included in the statistics, the total percentage infection would be considerably lower.

With regard to sexes, it is unfortunate that so few women can be examined, as the percentage among the small number seen appears to be high, and more cases would be of interest. The women, as a rule lead a more sedentary life than the men, and the infection seems to be commoner among persons of sedentary habits than those engaged in active pursuits.

The following table gives the occupations followed by the infected people of this series :—

Cultivator	45
Shopkeeper (including tailors, shoe makers, craftsmen, etc) ...	16
Housework	14
Boatmen and fishermen ...	14
Porter	5
Trader	4
Matmaker	4
Clerk	3
Other work	7

In concluding this paper I wish to express my thanks to Dr. G. C. Low, of the London School of Tropical Medicine, and to Dr. P. H. Ross, Government Bacteriologist, East Africa Protectorate, for valuable assistance and advice in the identification of the specimens of embryonic filariæ, and to Colonel A. Alcock, I.M.S., for kindly identifying specimens of mosquitoes for me.

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Addendum.—At a later date I was able to examine a series of cases in the Island of Pemba.

The people of Pemba are more primitive and conservative than any I met on the mainland.

The climate of the island is humid and unhealthy, and all forms of helminthic disease are very common.

Fifty specimens of blood were examined, all from natives of the island, and of these twenty showed infection with *Filaria bancrofti.* Some of these cases showed particularly large numbers of microfilariæ in the circulation.

A number of the patients were infected with ankylostomiasis as well, this disease being exceedingly prevalent in Pemba and Zanzibar.

The total of cases examined in Pemba and on the mainland amounts, therefore, to 367, with 132 showing filarial infection, or 35·96 per cent., a figure which agrees very closely with that found for the Lamu area In both areas the percentages of elephantiasis cases, roughly estimated, similarly correspond.

I have to acknowledge with thanks the permission of the Secretary of State for the Colonies to publish this work, which was done while in the public service.

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A CASE OF BILHARZIASIS CURED BY TARTAR EMETIC ADMINISTERED INTRAVENOUSLY,

BY

L. C. D. HERMITTE, M.B., CH.B. (Edin.).

The following case of endemic hæmaturia has been cured by the intravenous administration of tartar emetic, in general practice, and is published in support of the excellent work done by CHRISTOPHERSON in Egypt and CAWSTON in South Africa.

The course of treatment followed here is different from those used by either of these workers, but approaches that of CAWSTON'S. It was arranged on the recommendation of Colonel MARSHALL, Lecturer in Tropical Diseases in Edinburgh University, whose advice I took before CAWSTON'S article was published in the *Lancet* (17th April, 1920).

In CAWSTON'S experience an average total of 12 grains is generally sufficient to effect a cure of the disease. In the present case 13½ grains were necessary before fresh eggs ceased to appear in the urine.

The course was as follows :—

	Half grain	every 2nd day	for	3	doses.
Then	One grain	,,	,,	4	,,
	Two grains	,,	,,	4	,,

The urine was centrifugalised and examined daily so that the progress of treatment was closely and carefully watched. Slides were prepared from the outset, and a collection preserved for a final comparison. It has been most interesting to follow the gradual transformation of the

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ova, and their gradual disappearance from the urine. Before and during the early days of the treatment, they were elliptical, pale yellowish in colour, possessed a fine transparent shell with a minute sharp thorn-like spike at the posterior extremity, and hatched easily in water. As the treatment progressed the ova gradually became shrunken, shrivelled, blackish in colour with their contents granular, and could not be made to hatch, however long they were left in water. The injections were stopped immediately fresh eggs ceased to appear in the urine. Blackened ova kept appearing after the cessation of the injections, at first fairly numerous, but gradually becoming fewer, until on the eighteenth day after the last injection no ova appeared in the urine.

The examination of the urine was continned from time to time, until two months had elapsed since the last injection, and no more ova were found.

Along with the bilharzial infection the patient had a severe complicating cystitis with much pus and *B. coli* in the urine. On the recommendation of Colonel MARSHALL, washing out of the bladder was avoided. It was treated by hexamine both during the tartar emetic treatment and after. The cystitis cleared up after two months' internal administration of hexamine. The urine is now clear and normal, and all the other symptoms have disappeared. I think we may look upon the patient as cured.

During the treatment there were practically no complications, except headache, and this was easily combatted by the routine administration of aspirin, of which 10 grains were given two hours after each injection and 10 grains at bed-time when necessary. There was no call for the use of codeine, as recommended by CAWSTON, although towards the end of the treatment, when two grains were being administered, the patient complained of a little nausea and tightness of the chest. This was relieved by aspirin. Within half an hour of the 2-grain injections the patient had bouts of coughing which lasted on an average fifteen minutes.

History of the case.—The patient, G. N., was sent to Egypt with the 1st Welsh Horse, in January, 1916. He had no illness before he went out.

In February, 1917, after having been in Egypt over a year, he noticed

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that he had pain on micturition, and that his urine contained a large amount of thick blood.

He was transferred from Egypt on May 1st, 1918, to France, where he was admitted to hospital at Abbeville. There his urine was examined and for the first time his condition was diagnosed as bilharzial disease. He was admitted to hospital at Dewsbury, where he remained without treatment until the 25th August, 1918, when he was discharged.

He was admittted to the Burton-on-the-Water Cottage Hospital on April 17th, 1920, when a large number of pale yellow thin shelled ova, with terminal spikes were found in the urine.

The ova hatched easily after a few minutes in water; the hatching occurred more rapidly when warm water was used.

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RECORD OF TREATMENT OF INTRAVENOUS INJECTIONS OF TARTAR EMETIC.

Besides other Treatments mentioned below the Patient on the outset was put on Hexamine, gr. X., t.d.s. Diet ordinary throughout

		The Urine (Second 24-hour Specimens).							
Date.	Antimony Tartarate.	Reaction.	Deposit.	Blood.	Pus Cells.	Epithelial Cells.	Bacteria.	Crystals.	Ova.
April 21	Gr. ½ at 5 p.m.	Acid	Copious Pus, Blood Clots	+	+ +	—	B. Coli +	Few. Calcium Oxalate	Copious, all normal and easily hatched.
,, 22	—								
,, 23	Gr. ½ at 4 p.m.	Faintly Acid	Copious Pus	+	+ +	+	B. Coli + + +	Stellar Phosphate	Ditto.
,, 24	—								
,, 25	Gr. ½ at 2.45 p.m.	Ditto	Copious Pus	+	+ +	+	B. Coli + + +	Oxalates, Urates	Ditto.
,, 26	—								
,, 27	Gr. 1 at 2.30 p.m.	Ditto	Copious Pus, Crystals	—	+	—	B. Coli +	Stellar Phosphates	Ditto.
,, 28	—								
,, 29	Gr. 1 at 2.15 p.m.	Acid	Less Pus	—	+	+	B. Coli +	Triple Phosphate Oxalate	Copious, normal hatch after long standing in water.
,, 30	—								
May 1	Gr. 1 at 3.30 p.m.	Ditto	Less Pus, Phosphates	—	+	+	—	Stellar Phosphate	Copious, normal, one or two granular in appearance.
,, 2	—								
,, 3	Gr. 1 at 2.30 p.m.	Ditto	Less, Little Pus, Phosphates	—	+	+	+	Triple Phosphate and Urate	Very few normal, Ova all pale yellow.
,, 4	—								
,, 5	Gr. 2 at 5.30 p.m.	Ditto	Less Phosphates	—	+	+	+	Triple Phosphates	Most Ova still pale in colour, but more granular; a few much darker.
,, 6	—								
,, 7	Gr. 2 at 3 p.m.	Ditto	Scanty, Little Mucus	—	Very Few	+	+	Ca. Oxalate (large amt.)	Few Ova, pale granular; one or two darker.
,, 8	—								
,, 9	Gr. 2 at 5 p.m.	Ditto	Scanty	—	Very Few	+	+	Ca. Oxalate, Few Triple Phosphates	Mostly granular, darker and shrivelled.
,, 10	—								
,, 11	Gr. 2 at 4 p.m.	Ditto	Much Less	—	More	+	+	Ca. Oxalate	All granular and shrivelled. Fairly numerous.
,, 12	—								
,, 13	—	—	—	—	—	—	—	—	All Ova shrivelled, granular and black.

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Obituary.

SIR JOHN KIRK, G.C.M.G., K.C.B., M.D., F.R.S.

Honorary Fellow of the Royal Society of Tropical Medicine and Hygiene, 1914–1922.

Sir JOHN KIRK, whose death at Sevenoaks was announced on 15th January, 1922, at the age of 89 years, was an Honorary Fellow of the Society. His life takes us back to the days of Livingstone, whose chief colleague he was in the Zambesi expedition of 1858. KIRK was a son of the manse, born at Barry, Forfarshire, N.B., and as a boy was interested in botany and natural history generally.

After graduating at Edinburgh in 1854, he gained experience as a leader of a voluntary party of medical men who went out to the Dardanelles during the Crimean War, where he was assistant physician at a base hospital. Doubtless, he gained a knowledge of tropical disease in that campaign, and in 1858 he was attached to Livingstone's Zambesi party, primarily as a naturalist. He did valuable work as an explorer and scientist during five years, and was then appointed as Medical Officer at Zanzibar in 1866. It was, however, as an administrator that he did his best work, and he was first Assistant Consul, then Consul-General and Resident at Zanzibar, where he rendered yeoman service in upholding his country's interests, and in safeguarding the destinies of that great land.

Decorations and honours were showered upon him in recognition of his scientific and administrative powers. These included a K.C.M.G. in 1881, G.C.M.G. in 1886, and K.C.B. in 1890. In 1882 he received the Patron's Medal of the Geographical Society, of which for many years he was Foreign Secretary. He was an Hon. LL.D. (Edin.), D.Sc. (Cantab.), and D.C.L. (Oxon.), and was F.R.S. and F.L.S.

He took a prominent share in the opening up of East Africa; a worthy pioneer in whose steps a distinguished band of medical workers have followed, to Africa's lasting benefit. C. F. H.

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TRANSACTIONS

OF THE

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

JANUARY 19TH, 1922.

VOLUME XV. No. 7.

Proceedings of a meeting of the Society held on Thursday, 19th January, 1922, at 8.15 p.m., at 11, Chandos Street, Cavendish Square, W. 1, preceded by a Demonstration at 7.45 p.m. on "Some Stages in the Development of *Trypanosoma lewisi* of the Rat, by Dr. A. C. STEVENSON.

Sir JAMES CANTLIE, K.B.E., F.R.C.S. *(President)*, in the Chair.

SOME RELATIONS BETWEEN VEGETABLE AND HUMAN PATHOLOGY.

BY

E. J. BUTLER, C.I.E., M.B., *Director of the Imperial Bureau of Mycology.*

The differences in the organisation of the higher plants and animals are such—especially in the absence of true circulatory, nervous and digestive systems in plants—as to preclude any comparison between the great mass of the disorders from which the two kingdoms suffer. But since, in their ultimate structure, they are composed of the same cellular units, and since they are exposed to the attacks of similar parasites, it would be strange if there were no points of contact between vegetable and animal pathologists.

Vegetable pathology is a younger science than that of man and the higher animals, and there are, as yet, no handbooks that give in any detail a comparative survey of the pathological processes in plants.

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But, I think, there are grounds for hoping that the older science may yet gain something from the study of certain disease phenomena in plants. The directions in which this may be sought are in certain branches of cellular pathology; in the diseases involving changes in physiological processes, such as altered metabolism; in certain classes of parasitic and virus diseases; and in the effect of environmental conditions on the interaction of host and parasite.

I propose to give only a very few instances of disease processes in plants that have some analogy to those in the animal kingdom, but without attempting to define their exact bearing on particular aspects of human pathology. While they are not specially confined to the tropics they are prevalent there, and my own actual experience has been almost entirely tropical.

The first is tumour formation in plants, as exhibited by the well-known crown gall tumour caused by *Bacterium tumefaciens*. I have recently had an opportunity of going over with Dr. ERWIN F. SMITH, of Washington, the work on this disease on which he has been engaged for the last eighteen years. It is regarded by plant pathologists, the world over, as one of the masterpieces of scientific work in their field, and has raised Dr. SMITH to the position of a recognised leader amongst them.

The tumours produced by this organism on a multitude of plants are usually hyperplasia caused by the repeated division of the cells to produce a solid small-celled tumour tissue without cavities. The cells are often markedly embryonic in character, and divide mitotically and amitotically. Secondary tumours frequently develop, often at a considerable distance and deep-seated in the tissues. They are not caused by migration of the bacteria across normal tissues, but by a definite outgrowth from the primary tumour in the form of a tumour-strand of infected cells of marked characters, and easily recognised in section, though often only a few cells thick. Such strands have been traced for ten inches in length. The secondary tumours have the characters of the primary, so that if a leaf bears tumours from one on the stem, their structure will be a stem structure. Secondary tumours can also be produced by grafting a part of a primary tumour on a suitable part of the plant. Growth of the tumour is unlimited and devoid of polarity, and the destructive action on the plant is usually merely the result of pressure and crushing, though necrosis permitting secondary infection is common.

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The structure of the tumour varies according to the tissue primarily infected. Most of them are of the ordinary ground or conjunctive tissue cells, *e.g.*, in the cortex or pith, and are the nearest approach that one could expect to get in plants to Sarcomata. There is no epithelium in plants, and the epidermal tissues are thin, usually a single layer, and hard to inoculate without involving the underlying ground tissue. But a few cases have been described in which the tumour appears to be composed mainly of epidermal elements, including hairs (the plant hair being an outgrowth from a single epidermal cell), and these may be compared with Epitheliomata. The most interesting type, however, is Embryomata. These were first detected as recently as 1916. They are composed of a jumbled mass of young shoots and roots of usually incomplete structure, having only bits of organs, but sometimes forming more or less complete dwarf shoots. The secondary tumours developed from these have usually the same embryonic teratoma structure.

The organism which causes crown gall is a cell parasite of a highly developed type, in which the host cell is not destroyed but is stimulated to increased activity. There are many such cases in plant parasites. To the best of its ability the parasite aims at preserving the life of the host cell for its own needs. That it over-stimulates the cells, causing death by crushing and necrosis, may be regarded, more or less, as an accident.

Bacterium tumefaciens can be isolated and cultivated so that its morphology and characters are well known. It is rare in the tissues, at least in an active state; in one case it was estimated that there were only 200 living bacteria per cubic centimetre of the tumour. But perhaps the most significant thing that I learned from Dr. SMITH was that, contrary to his earlier belief, he is not now certain that he has ever recognised the organism in the tissue. What he previously saw he now thinks were only cell inclusions or chondriomes. Thus we have a parasite capable of causing a tumour having all the characters of a malignant growth; the organism has been isolated, grown in pure culture, and new tumours produced at will with it, but it is not with certainty to be recognised in the tissues of the tumour.

More recently Dr. SMITH has been able to produce experimentally small tumours of limited growth, having the characters of incipient crown galls, by injecting in suitable parts of plants certain of the diffusible products of the metabolism of *Bacterium tumefaciens*, including ammonia, amines,

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organic acids, etc., and he considers that the tumour is ordinarily caused by the continual production of small quantities of these excreted products by the parasite.

I am aware that the path of cancer research is strewn with the wrecks of parasitic theories; but the recent work on plant cancers, the experimental production of embryonic teratomata, the extreme difficulty of detecting the parasite in the cells (it is certainly *in*, not between the cells), and the evidence that it acts by the production of repeated small quantities of an excreted stimulus, makes it easy to understand the view held by Dr. SMITH, and other vegetable pathologists, that cancer will ultimately be found to be due to some similar cause.

A second direction in which disease phenomena in plants may throw light on certain animal diseases is found amongst the obscure group of diseases which the vegetable pathologists are calling "virus" or "contagium" diseases. These are diseases in which etiological studies suggest the presence of parasitic agent, but in which, with the means at our disposal, no parasite can be detected, and filtration through the Berkefeld filter does not cause a reduction in virulence.

The number of such diseases in plants is large, but many of them have only recently been recognised. They have all certain characters in common, but they fall into several groups, the most important of which is that known as the "mosaic" group, because its most obvious symptom is a variegated appearance of light and dark green patches on the leaves. So rapidly have new cases of mosaic been recorded in the last few years that one is tempted to say that every cultivated plant has at least one such disease. Cross-inoculations show that while allied plants (as, for instance, potato and tomato) may suffer from the same mosaic, the mosaics of distantly related plants are usually distinct.

These diseases are constitutional, the virus reaching all parts of the plant in three to four days after inoculation in the mosaic of cucumber. The incubation period before the first symptoms appear in this mosaic varies from four to fifteen days, and in potato and cabbage mosaic from twenty to thirty days. In sugar-cane mosaic the virus is restricted in its distribution, and may not reach every shoot. Many tissues and organs, though containing the virus, do not react to it in any way that has been hitherto detected. Also "carriers" have been found, *i.e.*, plants that show no symptoms after inoculation, yet contain infective virus in a

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transmissible form. Actual tissue reactions (cell modifications) are often caused in the leaves and fruit. Necrosis of the light areas in the leaves also occurs not uncommonly in the older stages. The other symptoms are due to deep-seated alterations in the physiological activities of cells and organs, revealing themselves in altered metabolism and abnormalities of growth (dwarfing, crinkling or folding of flat surfaces, shortening of axes, and, at times, proliferation). The disease is not usually fatal in annual plants, though death from weakness may occur. The reduction in vigour may be very great or slight. Mosaic and allied diseases of potato in England may cause a reduction of over 50 per cent. in the yield, and in Bermuda losses up to 75 per cent. have been recorded. On the other hand, I recently saw fields of sugar-cane in Louisiana with 100 per cent. infection from mosaic, but was informed by the local plant pathologist, EDGERTON, that the loss of yield was slight, though the disease has been present for seven years, and in other localities, *e.g.*, Hawaii, has been known to reduce the sugar yield by 60 per cent. The so-called deterioration of many cultivated plants, such as potatoes, leading to the constant demand for new varieties, has recently been recognised to be, in many cases, the result of the gradual accumulation of some one or other of these virus diseases.

From the standpoint of comparative pathology, what is important is the knowledge that has already been gained as to the methods of transmission and certain other characteristics of these virus diseases.

So far as is known all these diseases are insect borne. There are other methods by which infection can be produced, but the normal method in nature is by insects, chiefly sucking insects, especially aphids. In no case has transmission by mere contact been demonstrated. In one case, curly top of beet, no other method of infection than through the insect (a leaf hopper, *Eutettix*) has been discovered, and in this case the insect has to be kept for a period of twenty-four to forty-eight hours after feeding on a diseased plant before it can inoculate a healthy one, after which it can remain infective for 100 days. This suggests that a part of the life-cycle of the parasite is passed in the insect. In most cases, however, the virus can be obtained by extracting the juice (cell sap) of a diseased plant, and can be successfully inoculated on a healthy plant by injection into any part of the plant (tobacco mosaic) or into certain parts only (growing point in sugar-cane mosaic, any part but the root or flower

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in cucumber). It can also be transmitted often by crushing a diseased leaf on a healthy one, so as to injure both tissues, or even (cucumber mosaic) by bringing the broken hairs of healthy and diseased leaves into contact. In such cases the virus is evidently in each individual cell. In spinach blight the crushed bodies of aphids that have fed on diseased plants are infective. In this disease, too, the offspring of infected aphids are infective occasionally. In certain cases the disease is carried in the seed borne by diseased plants, but in a good many this does not occur. There is much evidence that the virus travels in the plant through that part of the vascular system, the phloem, in which the elaborated sap is conveyed. This system does not reach to the seed, the plant embryo feeding entirely by osmosis. It is most interesting to note that recent studies show that certain aphids, including some that are known to carry mosaic, feed by inserting their very fine and flexible probosces into the cells of the phloem, avoiding other cells as a rule.

One other method of transmission must be mentioned. Even in the most refractory cases, as beet curly top, infection can be secured by grafting a diseased on to a healthy plant, provided organic union is obtained.

After extraction from the plant, the life of the virus varies from only twenty-four hours (sometimes in cucumber mosaic) to a year or more (tobacco mosaic if dried). It is killed by a temperature usually of between 70°C. and 80°C., but can withstand exposure to very low temperatures. In some cases the ordinary antiseptics kill it, but the tobacco mosaic is resistant to most weak antiseptics. The concentration in the sap is such that a needle drop can be diluted to 1,000 without any loss of virulence, though virulence is reduced by a dilution to 10,000 (tobacco and cucumber). Filtration through porous filters depends on the size of the pore. The cucumber mosaic virus filters through the Berkefeld but not through the Chamberland; tobacco mosaic filters through both, but is retained by powdered talc.

Though there are still a few vegetable pathologists who prefer the enzymic or toxic hypotheses to the parasitic explanation of these diseases, most agree that their study has made it necessary to assume that they are caused by ultramicroscopic living organisms. The mere fact that they have not been successfully grown *in vitro* has no weight with the vegetable pathologist who is familiar with whole groups of parasitic fungi,

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such as the rusts, not one of which has ever been grown apart from its host plant. It is as certain as anything can be that no visible organism is present in extracted cell sap that is known to be highly infective. This does not preclude the possibility that the organism may have a visible stage, and indeed quite recently two independent workers have claimed to have discovered, in cells of sugar-cane and maize suffering from mosaic, bodies which in one case may perhaps be suggestive of a parasite not unlike a stage in *Rickettsia*.

Another set of problems, of no less interest to the vegetable than to the animal pathologist, is the geographical and seasonal distribution of certain diseases.

The experimental study of the relations of plants to their environment is naturally much easier than with human beings. In our botanic gardens we habitually grow tropical plants with success, being able to imitate their natural conditions. It is only recently, however, that plant pathologists have made use of their opportunities to study the influences of such climatic factors as humidity and temperature on plant disease. The results have been remarkable. It has been found possible to correlate the geographical distribution of a considerable group of plant diseases with the temperature and humidity reactions of the parasite and its host.

In the simplest cases a single factor, such as the degree of relative humidity of the air, acts by limiting the development of the parasite. One of the best examples of this is a disease of the rice plant which I studied in India. It was strictly confined to a large rice area at the head of the Bay of Bengal, and while the main crop from July to December was often totally destroyed, the secondary crop in the spring entirely escaped, though it was shown by artificial inoculations to be equally susceptible. I found that the organism could neither move nor feed nor reproduce at an air humidity of below 90°, while, though it is actively mobile, it is unable to feed or reproduce in water. To be able to multiply and attack the rice plant it therefore requires moderately prolonged periods when the air humidity is over 90°. This it only gets in certain limited areas in India during the second half of the year; and at a time when the disease was causing considerable alarm, it was possible to state that the bulk of the rice-growing area was not threatened.

The meteorologic factor may act by affecting the receptivity of the host-plant. Thus in the vine mildew, infection is dependent on the

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condition of the stomata, through which the parasite enters. If these remain semi-closed, as in young leaves or in dry conditions, infection is impossible. Infection has been found to fail if the moisture of the soil in which the vine is growing is below 15 per cent., unless the air humidity is above 80 per cent. If the soil moisture is above 20 per cent., the stomata open widely enough to permit infection at air humidities above 40 per cent.

But it would seem that in many cases the action of the meteorological factors on both the parasite and the host in their mutual relations—on what may be, for convenience, termed the host-parasite complex—has to be taken into account. In the onion smut, for instance, it is possible to get 100 per cent., or no successful infections, according to the soil and air temperatures used. The onion seed will germinate at soil temperatures from 10°C. to 30°C. but the best top growth is got with the soil temperature of 20°C. to 25°C. Infection by the smut fungus can be got freely from 10°C. to 25°C. but is slight at 27°C. At a soil and air temperature of 25°C. a great many plants will outgrow the infection and recover, but at an air temperature even as high as 30°C. to 33°C., infection can be got if the soil temperature is kept below 25°C. In outdoor plantings the disease was completely inhibited when the mean soil temperature reached 29°C., and increased progressively with lower temperatures. The relation is believed to act chiefly through the host-plant, as successful incipient infection can be got at the higher temperatures, but the subsequent development of the fungus is inhibited. The geographical distribution of the disease in the United States, where it is absent from large areas in which onions are sown in the warm season, has been completely explained by this work.

Recently it has been shown that the severe outbreaks of epidemic malaria in the Punjab are governed by a set of temperature and humidity factors affecting both the malarial parasite and the insect host. With a warm-blooded animal like man the temperature factor is probably of slight importance, except in so far as it acts on the parasite while exposed to the air, as in the external air-passages. But in insect-borne diseases, and in the life of the parasite outside its host, these studies have a certain interest, and the recent discovery that so small a difference as 5 per cent. relative humidity or 3°C. or 4°C. temperature can cause a difference of 100 per cent. in infection has come as a surprise to many plant pathologists.

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In these few instances, selected from widely different fields, I have tried to show that the problems which face the vegetable pathologist are often akin to those of human pathology. Where there is any evidence that disease processes are fundamentally alike in plants and animals, I venture to think that they are worth careful examination by pathologists generally. Their study in plants has many obvious advantages in the ease of experimentation and manipulation.

Vegetable pathology is under a great obligation to the older science, from which many of its methods and principles are borrowed, and there could be no more fitting way of repaying this debt than by the discovery that the study of plant diseases may throw light on some of the more obscure problems of human pathology.

Discussion.

Dr. A. Balfour: I think we must all welcome this paper very much. It is rather out of the usual run of papers which are given here, but it is the more interesting and instructive on that very account.

There are two matters to which I would like to make brief reference. The first, which is only indirectly associated with Dr. Butler's paper, is one which I know Sir Patrick Manson has had in his mind for many years, though I think he has never written about it. He is not here, but I do not think he will object to my mentioning the subject. It is the question, Why do certain plants produce alkaloids? Many of these are very poisonous substances; some are distinctly disagreeable to the taste. Sir Patrick asked me whether I thought these alkaloids were developed for the protection of the plants, for instance, against insect attacks. In cinchona bark there are, of course, quinine and other bitter alkaloids, and it would be interesting to know if those particular plants are subject to insect-borne diseases.*

The other point has a definite relation to the subject of the paper, namely, the production of gum by plants—a matter of great importance to those living in the Sudan, where gum acacia is one of the staple products. This production of gum is a diseased condition common to certain species of acacia tree, when they occur in dry regions and do not receive sufficient moisture. When the plants are well supplied with water this

* The interesting question of the function of alkaloids in plants is discussed by Annett in his "Investigations on Indian Opium," No. 2, in the *Memoirs of the Department of Agriculture in India*, Vol. VI., No. 2, Sept., 1921.—A. B.

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gum is not produced. In Australia, the work of GREIG SMITH showed that bacterial infection, in the case of two species of acacia, was responsible for the production of gum from the sugars of the sap. Later, RUHLAND isolated from diseased cherry trees an organism, now called *Bacillus spongivans*, which he cultivated, and which he found produced slime and gums.

Certain of the Ambrosia beetles have been incriminated by ZIMMERMAN as a cause of gum production, owing to the injury produced by their boring habits. In the Sudan, EDIE isolated a bi-polar staining organism from two species of acacia, and he cultivated this in a liquid medium containing levulose, glycerine, asparagin and potassium citrate; when so cultivated the organism produced slime and, eventually, gum. These are all very interesting observations, because it was definitely shown that gum is only formed when the tree is damaged, and a point of inoculation provided for the causative organism. EDIE proved that flies were operative, probably by conveying the bacterium in the gum from one tree to another. It is also possible that ants may be effective. I think this gum production may be a protective action on the part of the plant, and may possibly, in some cases, be analogous to the scab which forms over a wound and, to a certain extent, protects the latter from insects, and from invasion by micro-organisms.

Dr. P. H. MANSON-BAHR: We have evidence of *Bacillus tumefaciens* causing tumours in plants, yet the bacilli could not demonstrate in the tissues. This is a well-marked exception to the second of KOCH'S postulates. I think this should give us a lead in our further study of human pathology. In such a disease as sprue it may be possible for an intestinal bacillus to be responsible for the pathological condition, yet that bacillus, though it may be isolated by culture, may not be demonstrable in sections of the gut itself.

Dr. T. P. BEDDOES: I would like to ask to what extent there has been any treatment. When it comes to management of woodlands, certainly an improvement can be made. Take, for instance, one of the most destructive diseases—the larch disease. Instead of growing the larch unprotected in the open, if it is grown among other trees, such as the oak, then it grows in better condition. By management, woodlands can be very much improved in the matter of protection from diseases.

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Another question of interest is as to how far lime and manure affect treatment. There is nothing more interesting than to consider the use of lime. It was first introduced by the Romans, and, for many years, England was the centre of the lime trade, the substance being exported to Holland and the Lowlands. Then the industry went out for many years, and at about the middle of the eighteenth century in it came again, and certainly with very great improvement. Thus I am prompted to ask to what extent management—*i.e.*, the promotion of nourishment and similar factors—lessens the injurious conditions which are being discussed.

Dr. J. C. G. LEDINGHAM commented, as a welcome sign of the times, on the increasing evidence of helpful co-operation in problems of common interest between pathologists and bacteriologists in all spheres of microbiology.

The following also took part in the discussion:—Dr. C. F. HARFORD, Mr. A. D. COTTON, Mr. A. L. SHEATHER, Sir PERCY BASSETT-SMITH, and Dr. C. M. WENYON.

The PRESIDENT: We sincerely thank you, Dr. BUTLER, for having taken us over this new ground. I think plant pathology will find a place in the ordinary medical curriculum in the Tropical School, though this new subject may be thought by some to be making confusion worse confounded.

We are very much indebted to Dr. BUTLER and the various gentlemen who have spoken and brought up points of interest; and if each contributes his particular knowledge to the discussion going forward, we shall all learn a great deal.

Dr. E. J. BUTLER: Recently some work has been done in France on the group of so-called alkaloid producing plants, so as to study such substances as nicotine and atropine. In some cases, such as the aconite, it was shown they were definitely resistant to parasites if in concentration. Nicotine was not of sufficiently high concentration in the plant to have an effect on the organism. Certainly, many plants do successfully resist infection, because they have alkaloid in them, though whether the action is definitely protective is more doubtful.

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GREIG SMITH has shown gum production to be due to bacteria in a certain group of cases, but, in the commonest kind in this country, a parasite has not been detected; it is apparently a gummy degeneration of the cell wall, and the conditions which bring it out are a rapid alteration in the degree of humidity of the surrounding air. There is a sugar form to which Dr. GREIG SMITH has drawn attention. So far as I know these gums are not protective. and I am speaking of the acacias.

So far as I am aware, no work has been done in the way of conferring protection. In a few cases it has been said that products have conferred immunity to plants, but on careful examination every supposed instance has broken down.

I believe the transmission experiments to which I have referred have been done only on plants of the same species. I think Dr. SMITH has worked that out, but I cannot say definitely.

Replying to Dr. MANSON-BAHR, in artificial inoculation there is no difficulty in recovering the organism from the plant, though it cannot be seen there. Dr. SMITH has repeatedly cultivated the organism after the disease has been produced, though he could not see it while it was in the tissues.

In answer to Dr. BEDDOES, much work has been done on the control of these diseases, but it would take too long now to speak of it in any detail. The tree disease has been shown to be associated with attacks of certain large insects, and if you can keep the wounds down which are so produced, you improve the trees very much. Larch disease is said to occur more on certain soils than on others, and that is so with other groups of plants. There are plants which suffer from having excessive lime, and others which suffer from an insufficiency.

A paper was read by Dr. W. SALISBURY SHARPE, M.R.C.P., F.R.C.S., on "Diseases of the Ear in Relation to Life in the Tropics." This has been published in the *Journal of Tropical Medicine and Hygiene*, February 15th, 1922.

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COMMUNICATIONS.*

INFECTIOUS ABORTION OF CATTLE AND ITS POSSIBLE RELATION TO HUMAN HEALTH.

BY

L. E. W. BEVAN, M.R.C.V.S., *Government Veterinary Bacteriologist, Southern Rhodesia.*

[Delivered before the Southern Rhodesia Veterinary Association, Salisbury, 12th November, 1921.]

This meeting of the Southern Rhodesia Veterinary Association has been convened in order that its members may again discuss the very serious disease of cattle known as infectious, contagious or epizootic abortion, which since our last meeting has assumed alarming proportions in this country ; and in view of the recent suggestion that the disease may be infective to man, the members of the medical profession have been invited to assist in our deliberations.

The close association and interdependence of our two professions are daily becoming more noticeable, especially in tropical and sub-tropical countries where the diseases of man and animals are so closely allied. Undoubtedly the best and most scientific manner of studying such diseases is from the comparative standpoint, and the subject of my paper to-night affords an example of the necessity for co-operation between us.

For my part I have to bring before you certain aspects of the disease as it occurs amongst animals in this territory, but in the course of my remarks I may throw out hints which it is hoped may prompt the members of our sister profession to enter into the debate, and to bring forward any information they possess having a bearing upon the disease as it affects the human population of this colony.

There is no doubt that the future prosperity of Rhodesia principally depends upon the progress of the pastoral industry, but it is sad to relate that since the advent of the white man, the herds of this country have

*With a view to making the TRANSACTIONS more interesting, it is proposed to insert SHORT COMMUNICATIONS from Fellows relative to any matters of medical, sanitary or parasitological interest. It is felt that many Fellows in various parts of the world must be in a position to contribute short papers on these subjects, and the Secretaries will be pleased to receive any such from them.

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twice been decimated by bovine scourges. On the arrival of the pioneers it is said that the country was "rolling with cattle," but in 1896 the greater number of them were destroyed by rinderpest, which passed through this country on its way to the south. Notwithstanding this disaster the survivors, in the words of the Bible, brought forth their thousands and tens of thousands, until the industry was again upon a sound footing, only to be again destroyed by East Coast fever, which arrived in 1901, and has only recently yielded to the scientific efforts of the Veterinary Department.

At the present time it is officially stated that there are about one and a half million head of cattle in the country, owned equally by black and white, but, alas, the industry is again threatened with a new disaster in the appearance of this insidious and costly malady known as infectious abortion, a disease which is now known to exist in all parts of the territory.

The first outbreak recorded in this country was in a dairy herd in Salisbury, in 1906. This was promptly and vigorously dealt with, and was apparently stamped out. Although abortions undoubtedly continued to occur, they were not reported to the Veterinary Department, and it was impossible to determine whether infectious abortion as a specific disease was the cause of them until 1914, when the agglutination test was first applied as an aid to diagnosis. Since then it has been found to exist from Umtali in the east, through Rusapi, Marandellas, the Beatrice District, Enkeldoorn, Umvuna, Gwelo, Shangani, Bulawayo, and north to the Lomagundi District. In fact it is probable that it exists in cattle belonging to Europeans and natives alike throughout the country. It is equally prevalent in Northern Rhodesia, and, as far as one can gather, throughout the Union of South Africa.

This disease is not confined to Africa. In Great Britain it is second only in importance to and probably more prevalent than tuberculosis. In America it is stated by the Bureau of Animal Industry to be the cause of the loss of many millions of dollars yearly through its presence in dairy and range cattle. In New Zealand the losses in dairy products directly and indirectly attributable to it are estimated at £300,000 per annum.

In many ways this disease affects the human population of this country. The last census revealed a somewhat lamentable state of affairs

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and a population of little more than 33,000 white people. A great proportion of these are dependent upon the cattle industry, and I doubt very much whether, after his sad experience in the past, the Rhodesian settler, however stalwart he may be, can "stand up" to another disaster. And if it is found that this disease of cattle is infective to human beings, the situation is fraught with grave dangers, and we, the guardians of the public health, are faced with a grave responsibility if we wish to see Rhodesia, our adopted country of which we are so proud, survive the storm.

It has long been known that abortions, even in such numbers as to suggest some common cause, were apt to occur among domestic animals, and various reasons were advanced to account for them, as, for example, feeding upon ergotised pasture, sewage poisoning, mechanical injury, over-exertion, fright or sympathy; but as long ago as the beginning of the last century keen observers maintained that "an infection was the causative agent." The correctness of this view was proved later in the century, when abortion was artificially produced by the introduction of vaginal discharge and fœtal membranes into the vaginas of healthy cows. It was not, however, until 1896 that BANG and STRIBOLT demonstrated the specific cause to be the bacterium known as the *Bacillus abortus* of BANG, and, having cultivated this organism, in bringing about abortion by the injection of pure cultures into the vagina of pregnant cows and ewes. Also injections of cultures into the circulation produced similar results. Later, MCFADYEAN and STOCKMAN demonstrated what is of still greater practical importance, that pregnant animals could be infected by feeding upon infective material, either artificial cultures or natural exudates from infected animals. In these cases artifically produced, the characteristic lesions of the disease were met with, and the bacillus could be recovered from the exudate of the fœtal membranes, the diseased cotyledons, and the intestinal contents of the aborted fœtus.

It is probable that the organism had been noted by previous observers, but it remained with BANG to isolate the bacillus, which required for primary cultures not only a specific medium, consisting of nutrient agar to which liquid gelatin and sterile liquid blood serum had been added, but also an optimum growth temperature of 37° C., and a special oxygen content indicated by the fact that the colonies develop in a definite stratum of a few millimetres below the service. The

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B. abortus is thus not a strict anaerobe, but has been classified as a micro-aerophile.

Various devices have been made use of in order to establish a primary culture and to afford the special oxygen requirements. But it would appear that in subcultures the organism can accommodate itself to ordinary aerobic conditions. Under such conditions surface colonies develop in about three days as small, usually discrete honey-like dots. These later tend to coalesce and to form a creamy film, which in time becomes darker in colour, and in very old cultures is actually coffee-coloured or brown.

The medium upon which the strain used in our laboratory, and to which the organism appears to have become well accommodated, is prepared as follows:—

Lemco	5	gm.
Sodium chloride	5	gm.
Peptons	10	gm.
Agar	27½	gm.
Water	1	litre.
Glycerine	3	per cent.

added to the filtered medium which has previously been rendered alkaline.

Surface growths upon this medium are well established in three days, and fully developed growths suitable for vaccines are available within a week. A certain degree of moisture favours the growth, and for vaccines it has been found necessary to conserve in a special manner the water of condensation. Too much moisture, however, alters the cultural characteristics of the growth and the properties of the organism.

The *B. abortus*, both in natural and artificial conditions, tends to collect in clusters, and these are to be met with in the affected chorion, cotyledons, characterised exudate and discharges, and in the stomach of the fœtus. They are stained readily by basic aniline dyes, but in the clusters the morphology is difficult to define. The term coccobacillus has been used to describe the organism, from the fact that coccoid and elongated coccoid or bacillary types are met with.

Some difficulty is experienced in establishing primary cultures, chiefly on account of the peculiar oxygen requirements of the organism. Several ingenious methods have been devised to cope with this, for example,

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suspected material is sown on a medium, which is placed in a closed jar with other tubes containing common aerobic organisms, which exhaust the oxygen until the stage when the *B. abortus* finds the atmosphere congenial. This method is made use of to separate the *B. abortus* from other organisms with which it may be associated in septic discharges; but, perhaps, the best method of obtaining a pure culture is to inoculate suspected material into guinea-pigs, when the *B. abortus* becomes established in the spleen, which becomes enlarged, and sometimes, after two or three months, beset with tubercles. In a few weeks, however, the organism is present in the spleen in pure culture, and in such numbers that, when small pieces of spleen substance are spread upon agar slants, a characteristic growth can be obtained. This method has proved useful in demonstrating the presence of the specific micro-organism in the udders and milk of apparently healthy cows acting as carriers of infection.

In 1918, EVANS pointed out a close relationship between the organism responsible for infectious abortion of domesticated animals and the so-called *Micrococcus melitensis*, the cause of the well-known undulant or Malta fever in man. More recently MEYER and SHAW have stated (*Jl. Infect. Dis.*, 1920, Sept., Vol. 27. No. 3, pp. 170-184) that "on morphological grounds the organisms of undulant fever and infectious abortion of domesticated animals must, therefore, be considered as identical," and suggest that a genus, for which they propose the name "Brucella," be created in the family Bacteriaceæ, to separate and distinguish properly these important micro-organisms.

The serum of animals affected with infectious abortion will agglutinate the *B. abortus*, and, in 1914, BEVAN was able to demonstrate that the serum of cattle suffering from the disease in Southern Rhodesia would agglutinate strains of *B. abortus* obtained from Great Britain, the Union of South Africa, and British East Africa. Similarly, serum from natural cases of infection in British cattle would cause agglutination in emulsions of these strains. This observation suggests that the causal organism of bovine abortion in Southern Rhodesia is similar or identical to the *B. abortus* causing the disease in other parts of the world—a matter of some importance, as will be seen later. It is probable, however, that there are different strains of the organism of varying virulence, just as there are strains of the *M. melitensis*; and it is conceivable that there may be a certain overlapping of strains of the two organisms.

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In February last, a patient from the Shamva District was admitted to the Salisbury Hospital, and his serum was obtained by Dr. PEALL and tested by BEVAN against cultures of *B. abortus*, with which it caused rapid and marked agglutination in dilutions of 1 in 200. This, I believe, was the first demonstration of a co-relation between the abortion of cattle and undulant fever of man in this country. The test was prompted by an article by FEUSIER and MEYER, in which they showed by serological tests a close association between *B. abortus* and *M. melitensis*, and pointed out that certain strains of *M. melitensis* bore a closer relationship to the abortion bacillus than to other strains of melitensis. They also demonstrated that "the serums from cows and hogs suffering from natural abortion disease may also react to both *B. abortus* and *B. melitensis* organisms."

Recent observations by ORPEN, working in the laboratory of the Medical Department, and by BEVAN, working independently, reveal a very serious state of affairs in this country, if serological tests and cross-agglutination reactions are to be regarded as indicating any corresponding pathogenicity of the organisms.

The possibility of such an association has engaged the attention of numerous observers for many years past in other parts of the world, and in July, 1916, COOLEDGE, in the *Journal of Medical Research*, reviewed the experiments made up to that time by several observers. MOHLER and TRAUM obtained cultures from guinea-pigs inoculated from the tonsils and adenoids of fifty-six human beings, and obtained from one case distinct lesions of infection in the liver, spleen and testicles, from which the *B. abortus* was recovered.

LARSEN and SEDGWICK made an extensive study of human infection by this organism. They examined the blood of 425 children, and obtained 73 positive reactions by the complement-fixation method. They also found that an infant taken from the breast on the seventh day after birth and fed on cow's milk, gave a positive reaction on the twenty-first day. COOLEDGE himself examined the blood sera of 14 persons drinking raw milk, pasteurised milk, and no milk, and found that of the persons who had been taking raw milk or cream previous to the test, three (or 50 per cent.) gave a reaction to the complement-fixation and agglutination tests. One woman, whose blood reacted to the test, had been drinking milk which was found to contain *B. abortus* antibodies, and had been drawn

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from one quarter of a certain cow. One man, whose serum gave a partial agglutination at 1 in 100, had been drinking about a pint of raw cream daily during the summer. These results satisfied the author that the presence of antibodies in human sera is due to the ingestion of infected milk, and may indicate either active infection or merely a passive immunity due to the absorption of antibodies from the milk, and he adds "we have no proof that *B. abortus*, BANG, is pathogenic for human beings." No reference is made in these reports to any symptoms in these reactions suggesting undulant fever; nor are we told whether abortion at any time took place among human females fed upon infected milk.

It should be remembered that in natural conditions abortion due to this organism is essentially a disease of cattle, and it is stated by the Departmental Committee appointed in 1905 by the Board of Agriculture and Fisheries to enquire into epizootic abortion, that it had no hesitation in stating that it believed at least 99 per cent. of the outbreaks of cattle abortion which assumed epizootic characters, to be due to infection by the bacillus of cattle abortion.

Mares, sheep and swine may also become infected with the bacillus of BANG under natural conditions and by artificial methods. Goats, guinea-pigs and rabbits have also proved susceptible, and it may well be that the human female is not exempt.

Natural infection occurs by the vagina or by the mouth. It is probable that ingestion is the most common method. The bull is occasionally a mechanical transmitter of contagion, and it has been shown that the organism may become localised in the vesiculæ seminales, deferentia, testes and epididymides of the male, which may thus become a carrier of infection.

The symptoms in cattle may be briefly described. The disease is generally most severe in heifers and young cows, but may attack a susceptible animal of any age and at any stage of gestation. It is said that "the majority of cases of 'visible' abortion occur between the fifth and seventh month of gestation; the average period is between the fifth and sixth month, if a reliable estimate of the early or invisible abortions could be made. Some cows abort about the first or second month, and no suspicion is aroused until œstrum recurs a few days later" (AINSWORTH WILSON). The suspicion that animals may frequently abort unnoted is confirmed by our observations in the country.

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The course of the disease is insidious, and its existence is often unsuspected until the shortage of calves draws attention to its presence. In this country the diagnosis is confirmed by a special modification of the agglutination test.

The act of abortion is generally easy. Premonitory symptoms may or may not be present, and in many cases the fact that abortion has taken place is only detected by the discovery of the fœtus or by a peculiar yellow flocculent discharge which soils the tail and hind parts, or by the retention of the after-birth, which is a very frequent sequel. Particular attention may be drawn to the fact that there is no appreciable effect upon the animal's general health and condition; that is to say, febrile disturbance, anæmia, rheumatic pain or indications of neuritis have not been observed. Pathological changes are almost entirely confined to the uterus and to its contents. The mucous membrane may be in a state of catarrh and a typical exudate may cover the chorin. This is somewhat characteristic, being of a lightish yellow colour varying to dark brown. The membranes may be œdematous and the cotyledons may be swollen, and occasionally necrotic. The fœtus frequently appears quite normal; it is sometimes mummified, but is rarely putrid.

The disease may be regarded as a disease of the fœtal membranes. If the animal is not pregnant the organism can produce no pathogenic effects, and it is known that in apparently healthy animals the udder may be heavily charged with abortion bacilli, without harm to the animal until it becomes pregnant, when the organism may again make its way to the uterus. SCHROEDER and COTTON record "a number of calves produced by cows with infected udders were killed immediately after they were born, and their bodies tested for the presence of abortion bacilli through guinea-pig inoculation methods. It was found that such calves —those that were delivered alive and seemingly healthy and vigorous— may harbour bacilli in their stomachs and gastrohepatic lymph glands, but invariably, when the calves were infected, the after-birth and uteri of the dams were also infected." Animals that thus harbour the organisms in their udders are a constant source of danger—certainly to cattle, possibly to man. They act as carriers, and may be the means of infection of a perfectly clean herd, or of re-infection in a herd which in the course of time has become clean. So long as such animals are in lactation the bacilli may be excreted in the milk. If the human subject is liable to a

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disease caused by this organism, this source of infection is of considerable importance.

Let us now compare the symptoms of Malta fever in goats. The following description by DUBOIS is quoted by EDWARDS, in a most comprehensive paper read by him before the National Veterinary Medical Association, to which I am largely indebted for many of the details I have brought before you. DUBOIS writes, " . . . in the goat there are as a rule no visible symptoms, and the animals maintain to all appearance their good health, the onset of the disease always passing unnoticed . . . . The most important symptom, and the one that would always make one suspect Malta fever, is abortion, but even that is not constant, nor is it diagnostic. When the disease makes its appearance in a herd for the first time, the percentage of abortions may be anything from 50 to 90 per cent. of the pregnant goats. If these goats are again served, abortion occurs again, but the percentage is not so great as on the first occasion. Abortion may cease completely in a herd, but this does not make the danger any less, as a certain number of animals always harbour the parasite. Sterility is rarely observed." " In the sheep the symptoms shown closely resemble those in the goat, and, as in the goat, abortion is the most important symptom." " In all animals, independently of the clinical symptoms, the blood serum, urine, and milk possess agglutinating powers for *M. melitensis*. The value of the (agglutination) test (in diagnosis) is indisputable, but since the serum may retain its agglutinating power for years after all trace of infection has disappeared, a positive result only indicates that the animal is or has at one time been affected."

Thus we see that *M. melitensis* causes in certain of the lower animals a disease similar to that caused by *B. abortus* in cattle. But it also gives rise to a disease in man characterised by entirely different symptoms. In Rhodesia a number of cases have recently occurred among human beings who have shown the clinical manifestations of Malta fever, but, as far as can be ascertained, have not imbibed goat's milk, but have resided on farms where infectious abortion of cattle is known to exist. Circumstantial evidence, therefore, points to infection through infected cattle, but direct proof is not yet available.

The most powerful argument against the infection of man by the *B. abortus* of cattle is advanced by EDWARDS in the paper referred to.

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He says, " . . . there can be but little doubt that the two organisms (*i.e.*, *M. melitensis* and *B. abortus*) differ in the all-important property of pathogenicity, for otherwise, in view of what we know concerning the excretion of the abortion bacillus in the milk, and the large percentage of cows that act as carriers of the bacillus, a large part of the population of Northern Europe and the United States would be liable to infection, but evidence to hand has failed to prove that the drinking of milk from aborted cows is a source of danger to human beings." On the other hand, it has been stated that Malta fever does not originate north of latitude 46°, but cases have been observed in England by MANSON himself. It has been pointed out that Switzerland and South Germany are the home of the milch goat. Nevertheless, these countries are particularly free from Malta fever.

If *B. abortus*, even occasionally, caused in man, in other parts of the world where infection of cattle is common, the remarkable symptoms manifested by patients from the Marandellas District and elsewhere in this country, the fact must surely have been noted; or if the symptoms of Malta fever were common in countries where the drinking of goat's milk could be excluded as a possible source of infection, but where infectious abortion was known to prevail, the circumstance would scarcely have been overlooked. It is certainly remarkable that although specific abortion of cattle has been prevalent in the Marandellas District since 1914, it is only within the last few months that the so-called Malta fever cases in man have been detected.

The suspicion that abortion among human females might have been associated with the drinking of infected milk, however, has frequently been suspected and suggested.

It is also a fact worthy of consideration that on farms where men have been infected, the other members of the family have apparently escaped, although if infected milk is the cause of the disease, one would have expected the women and children to have been the chief victims.

In this connection it should be mentioned that the Malta fever organism is not a delicate organism, and is very resistant to desiccation. It is, therefore, probably blown about in dust. Moreover, it is excreted in the urine of apparently healthy goats and cows. Possibly the *B. abortus* is disseminated in a similar manner, and, if so, suspicion might fall upon the dust of cattle kraals where infected animals have been herded, which

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would explain the large proportion of cases among males as compared with women and children.

Another statement made by EDWARDS may be quoted. He says: "Experimentally it has been found quite easy to infect and cause abortion among other farm animals and in small laboratory animals by causing them to be inoculated with, or to ingest, doses of BANG'S bacillus, and it is quite possible that even man may become infected; but the natural reservoir of the disease-producing organism is the cow, and there is every reason to believe that just as the bovine type of tubercle bacillus would cease to exist if bovine tuberculosis were completely eradicated, so would BANG'S bacillus no longer persist if the bovine species became extinct. In devising control measures, therefore, other animals than cows need not engage our attention."

A grave responsibility is thus imposed upon my profession, and I may be forgiven if I give a brief outline of the measures which have been adopted by the Veterinary Department of this country, in its endeavour to cope with a disease which, up to the present, has defied the efforts of administrations in other parts of the world.

At a meeting of the professional officers of the Veterinary Department, held in the Veterinary Department, Salisbury, in August, 1920, to discuss this disease, it was decided that:—

> "Official quarantine of infected farms and herds was impracticable, and tended to defeat its own ends by leading to the suppression of information as to the existence of the disease. Similarly, the publication of notices on the doors of magistrates' courts and in the Government *Gazette* was not desirable. It was admitted that the only method at present available to the Veterinary Department of limiting the distribution and dissemination of the disease was in the power of veterinary officers to refuse permits for the removal of infected or suspected cattle.
>
> "It was decided that the best method of controlling the disease from the point of view of veterinary administration, at the present time, was to educate and assist the public, and it was suggested that veterinary officers should, when possible, deliver lectures on the subject in their respective districts.
>
> "It was agreed that the best method of detecting or determining the specific nature of any outbreak of abortion was by BEVAN'S

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modification of the pipette method of collecting blood for the agglutination test.

"The advisability of advising purchasers of cattle to buy 'subject to the test' was discussed, but it was not considered feasible. It was decided, however, that veterinary officers should warn and give advice to buyers contemplating purchase of animals likely to be infected.

"It was agreed that the reports received from veterinary officers and stock-owners as to the efficacy of the vaccine as prepared at the Veterinary Laboratory by the Bacteriologist were most satisfactory, and it was decided that the method should be recommended and applied when possible, that is, when supplies were available.

"The danger of the bull as a source of infection was discussed, and it was decided that all bulls in infected herds should be tested, and, if yielding a positive reaction to the agglutination test, should be destroyed, the test to be applied before vaccination. It was decided that non-reacting bulls should be removed from the herd for a period of six months, and should be vaccinated with the devitalised vaccine.

"It was decided that all female stock in an infected herd should be vaccinated with devitalised vaccine, including calves, and in this way an immune herd should be established. New stock should not be introduced to the herd until a normal calf crop indicated the immunity of the herd, and then only after inoculation.

"It was agreed that so-called 'queen' cows were a source of danger and should be tested, and, if reacting, should be destroyed.

"It was decided that vaccine should be issued through the district veterinary surgeons, in order that they might be aware of the existence of the disease in their districts, the first vaccination to be under the control of the veterinary officer in charge, but, if necessary, subsequent injections to be performed by the owner.

"It was thought necessary to point out to owners that the vaccination could not restore life to a dead fœtus, but might hasten the immunity of an animal which had aborted and prevent non-pregnant animals from becoming infected.

"It was pointed out that a considerable number of barren cows failed to react, and that these could only be destroyed at the owner's discretion. The cause of their condition was probably other than infection by the *B. abortus*.

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"It was decided that in all outbreaks an attempt should be made to trace the source and origin of the infection. It was considered desirable to apply the test when a number of cases of retention of after-birth was reported in a herd."

The special method of collecting blood for the agglutination test referred to is merely a modification of the WRIGHT capsule, used with a certain preservative mixture which is added to the blood to prevent decomposition. Hitherto, in this country and at the present time elsewhere, the technique of collection was crude and unsatisfactory. The animal was secured and generally thrown; a cord was passed round the base of the neck in order to "raise" the jugular vein, from which blood was drawn off into a sterile bottle. The operation had to be conducted under aseptic conditions, any extraneous organism making its way into the blood setting up changes which interfered with the accuracy of the test. The difficulties of successfully maintaining such conditions in veterinary practice may be readily understood by those having any field experience. The result was that at least 95 per cent. of specimens sent for test arrived in a putrid or hæmolysed state and quite unsuitable for diagnosis. The pipette originally devised was merely a piece of glass tubing with a 4 mm. bore, and drawn out to a point at each end. The animal having been secured and its ear damped and dried, a bold cut was made across the posterior auricular vein, so that the blood escaped freely. A small quantity of preservative having first been run into the pipette to about a quarter of its length, the point was applied to the blood accumulating at the wound, so that it was drawn into it by capillarity. The pipette was then sealed by sealing-wax or other adhesive material. By this method at least 95 per cent. of specimens submitted have arrived suitable for testing at the laboratory, and it may be claimed that this simple invention has been the means of detecting the disease in many localities where otherwise it might have been overlooked.

After many experiments a still more simple method has been devised, by means of which the veterinary officer, or indeed the farmer himself, will be able to test suspected animals without sending the material to the laboratory. The method may prove useful to medical practitioners, enabling them to carry out their own tests with *M. melitensis*, or indeed any micro-organism which will react to agglutinins.

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Reference is also made to the devitalised vaccine prepared at this laboratory. The term "devitalised" has been coined to distinguish the vaccine from that which is generally referred to as "dead" vaccine, in which the micro-organisms have been killed by heat.

Since 1914 STOCKMAN has applied a vaccine to virgin and barren cows, consisting of enormous doses of living bacilli. This apparently strange procedure is based upon a well-known belief in other countries, that the majority of cows infected with infectious abortion do not abort at the successive pregnancies, or that if a cow aborts a second time, it rarely aborts a third.

It is also a well-known peculiarity in connection with infectious abortion in other countries that, provided infected animals are not introduced from without, the disease tends to wear itself out, and in a few years to disappear from the herd. Also it is known that if a long interval, say four to six months, elapses between abortion and the next impregnation, the cow will carry her calf to full term. These observations suggested that in nature an immunity is sooner or later set up in a non-pregnant female at one time infected, and it was sought by means of the introduction of vaccines containing the living organism to imitate or hasten the natural process. The method, however, is fraught with danger, especially in this country, where it is often difficult to determine whether the cattle are in calf or not, and where primitive methods of transport render it unsafe to issue infective material.

A second method was tested, namely, the inoculation of cattle with vaccines made up of organisms destroyed by heat. STOCKMAN having drawn the deduction that the results did not justify the trouble involved, condemned the method; an example which has been followed by others, in spite of the fact that the conditions under which his tests were made were not free from objections. For example, the dead vaccine was inoculated into cows already pregnant, these cows being chosen from a badly infected herd, so that the fœtus of some of them may have been seriously damaged before the vaccine was applied. German experimenters (ZWICK, ZELLER, KRAGE and GRUNDER) have also found that living vaccine yields the best results, but even with dead vaccines figures show a well-marked reduction in cases. There is, however, in my profession, as in others, a tendency to accept without criticism the voice of the oracles. One grave disadvantage attaches to the living vaccine, namely, that even

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when applied to non-pregnant animals the organism may become localised in the udder, from whence, when the animal becomes pregnant, it may infect the fœtal membranes. These being discharged when the calf is born, may set up infection in a clean herd, or re-infection in a herd from which the disease has died out.

BEVAN has shown that the agglutination reaction produced by a heated "dead" vaccine, although not as strong as that produced by living vaccine, is nevertheless quite appreciable; and if agglutination may be taken as an index of the progress of immunity, the reaction set up should prove a valuable adjunct in dealing with this disease. But the heating of an organism to the temperature generally applied in producing a vaccine, coagulates the albumen of the organism and destroys its toxins. It also, as may be seen in the test tubes exhibited, renders the organism insusceptible to agglutinating sera, facts which may be seriously considered by the medical profession, who have to rely so often upon dead vaccines.

To overcome these objections, BEVAN devised a method of destroying the organisms employed in his vaccines by exposure to chloroform, which, although rendering the organism inert, does not destroy its toxins. A vaccine made in this way will still agglutinate, which cannot be said of many of the vaccines composed of organisms destroyed by heat.

Good results have followed the use of this vaccine when suitably applied, as shown by the reports received from district veterinary surgeons, but, in some instances, too much has been expected of it. In most cases where it has been pronounced a failure, careful investigation has shown that instructions were not carried out, or that many of the animals at the time of treatment were already well advanced in pregnancy, and probably infected, so that the expulsion of the calf was inevitable. In some cases also the dose was too small to render the animal immune throughout the nine months of pregnancy, and in future the quantity of organisms to the dose will be greatly increased, and the timing of the injections will have to be altered, so that the immunity conveyed will be maintained throughout the full period of pregnancy. Apart from its intrinsic value, the vaccine is a useful adjunct to administrative measures. The knowledge that animals can be treated has prompted owners of infected herds to report, and has justified the Veterinary Department in refusing permits for the movement of such herds during the three months' period of treatment.

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I think we may justly claim that the methods adopted by the Veterinary Department of this country in dealing with infectious abortion are far superior to any obtaining elsewhere. However imperfect they may be, an earnest endeavour has been made to cope with a disease which other administrations have admitted their inability to control, and have removed from the list of scheduled diseases a lamentable confession of inertia and impotence.

CONCLUSIONS.

1. The disease known as infectious abortion of cattle, which exists in Southern Rhodesia, is caused by the *B. abortus* of BANG, and serological tests indicate that it is identical with the specific abortion of cattle in other parts of the world.

2. The *B. abortus* of BANG bears a close morphological and biological resemblance to the *M. melitensis*. Serological tests also indicate a close affinity, in that certain strains of *M. melitensis* can be agglutinated with serum from cattle infected with infectious abortion, and strains of *B. abortus* can be agglutinated by serum from human patients suffering from undulant fever.

3. The characteristic symptom of undulant fever in goats and sheep is abortion.

4. Certain cases presenting symptoms resembling undulant fever have occurred in Southern Rhodesia in men who, as far as is known, have not obtained infection from goats, but have resided on farms where animals are or have been infected with infectious abortion.

5. It is suggested that these cases are suffering from an infection caused by the *B. abortus* of cattle.

6. It is also suggested that human females may contract infectious abortion through the ingestion of the organism in milk and dairy products from infected cows.

7. Although infectious abortion is prevalent in other parts of the world where goat's milk is not used for human consumption, it has not been accused of causing the symptoms of undulant fever in man, nor has it been proved to cause abortion in human subjects.

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8. Although infectious abortion has prevailed in the Marandellas District for many years, it is only within the last few months that cases resembling undulant fever in man have been recorded.

9. While circumstantial evidence points to the infection of man by the *B. abortus* of BANG, and scientific tests up to a point support this suspicion, the final proof is not yet available.

10. If infectious abortion of cattle is essentially a bovine disease, a grave responsibility attaches to the Veterinary Department; and if this disease is communicable to man, a hearty co-operation between the medical and veterinary professions is necessary in dealing with it.

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A SIMPLE CONTAINER FOR TRANSPORT OF SMALLPOX LYMPH IN THE TROPICS.

By H. LYNDHURST DUKE, B.A., B.C., M.D., *Bacteriologist, Entebbe, Uganda Protectorate.*

The following is a brief account of a simple carrier for smallpox lymph in the tropics. The container has been designed by Mr. E. C. Haddon, Laboratory Assistant, Uganda. It is of strong construction, and can be made quite cheaply in the bazaar.

Certain experiments have been performed to test the efficacy of the container, as a means of maintaining lymph at a lower temperature than that of the external air. Mr. Haddon's results, which were obtained with a crude model, show that, with water as the sole cooling agent, a marked difference is maintained between the temperature in and outside the apparatus, even under very adverse conditions of maintenance. This container will receive a further trial in Uganda, and it is hoped that this note will induce others to give a trial to this simple, strong, shock-absorbing and easily-made apparatus.

The experiments carried out are as follows:—

Test No. 1—20th October, 1921.

Conditions of Test.—The glass phial, marked *A* in the diagram, represents the receptacle in which the lymph, made at the Entebbe Laboratory, is at present transported. Under the present system of transport the phial is then dropped into a close-fitting, hollowed-out wooden block, padded at either end with a plug of wool, and the block enclosed in brown paper.

In Test No. 1, two similar phials are taken, and 40 c.c. of tap-water are placed in each in lieu of the actual lymph. One phial, *A*, is placed in the apparatus, in the position shown on diagram; the other—the control *C*—is dropped into its wooden block. Both apparatus and control are then placed on the ground, on a white pad of wool, in full glare of

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the sun. A thermometer was placed on the pad alongside. The advantage of the apparatus over the block is shown by the following readings :—

Time	External Temp. °C.	Temp. of A. °C.	Temp. of C. °C.	Ext. T.—Temp. of A. °C.
9.15 a.m.	31	22	22	9
10.0 a.m.	32	22·5	25·5	9·5
10.40 a.m.	31	24·5	30·5	6·5
11.30 a.m.	40	25	33	15
12.0 noon	37	25·5	35	11·5
1.30 p.m.	35	24	35	11
2.30 p.m.	33	25	35	8
3.30 p.m.	31	24·5	32·5	6·5
Apparatus removed to laboratory room and placed on the bench.				
4.0 p.m.	27	23	29	4
5.0 p.m.	24	20	24	4

Test No. 2—21st October, 1921.

Conditions of Test.—The apparatus had been left on the laboratory bench overnight, and the tests or observations were continued in the laboratory on the following day. The external temperature was obtained by laying the thermometer on the laboratory bench :—

Time.	External Temp. °C.	Temp. of A. °C.	Temp. of C. °C.	Ext. T.—Temp. of A. °C.
8.30 a.m.	24	20	24	4
10.0 a.m.	25	21·5	24·5	3·5
12.0 noon	26·5	22	25·5	4·5
1.30 p.m.	27	22·5	26·5	4·5
3.30 p.m.	27·5	23	27	4·5

Test No. 3—22nd October, 1921.

Conditions of Test.—Apparatus placed in incubator which contained a large open vessel of water :—

Time.	External Temp. °C.	Temp. of A. °C.	Temp. of C. °C.	Ext. T.—Temp. of A. °C.
8.30 a.m.	27 (room)	21·5	24	5·5
11.30 a.m.	36·5	31	35	5·5
2.30 p.m.	36·5	30·5	36	6

It will be noted that the tests were made under varying, and all of them severe, conditions, and that throughout the whole of the experiment an average difference of 5·8°C. was maintained. A noteworthy fact is that this average increases with rise of external temperature, so that the action of the apparatus appears to be compensatory, that is, its

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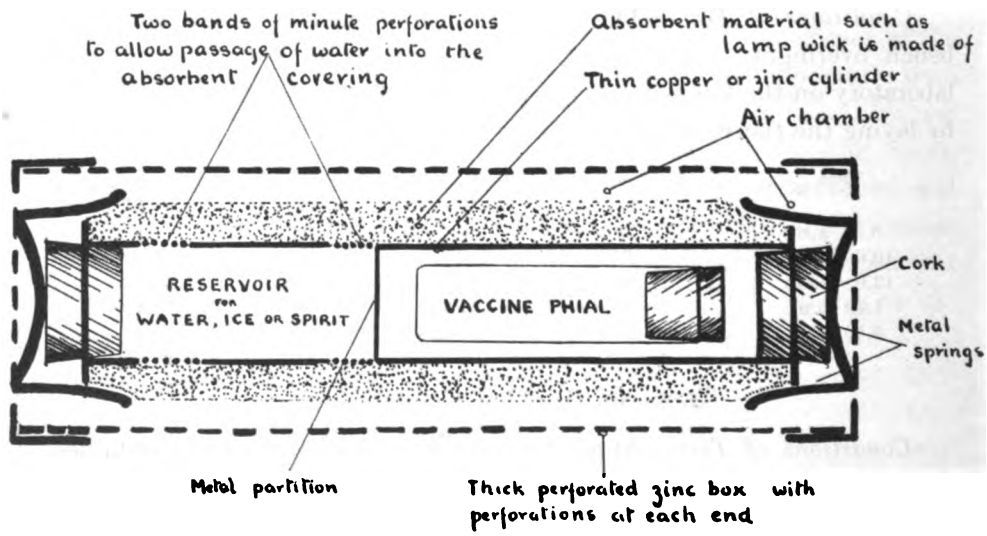
effectiveness increases as the external heat becomes greater. This is evidenced by the following table:—

External Temperature. °C.		Diff. maintained by Carrier. °C.		Other Conditions.
37	...	11·5	...	Open air direct to sun's rays.
36·5	...	6	...	Hot, humid atmosphere.
31·7	...	5·5	...	Ditto.
27	...	4·5	...	Warm, shady position.
25·5	...	4	...	Ditto.
25·5	...	3·5	...	Ditto.

The most severe test is undoubtedly No. 3, and it is to be noted that, even under such conditions, the carrier was able to maintain a difference in temperature of 5° C.



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TRANSACTIONS

OF THE

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

FEBRUARY 16TH, 1922.

VOLUME XV. No. 8.

DEATH OF SIR PATRICK MANSON, G.C.M.G., F.R.S.

As this number of the TRANSACTIONS went to Press, the sad news of Sir PATRICK MANSON's death on Sunday, April 9th, was received. On behalf of the Society, the Secretaries sent a wreath with an appropriate note to Lady MANSON. At the Memorial Service, held in St. Paul's Cathedral, on April 12th, the Society was represented by the President, Sir JAMES CANTLIE, K.B.E.

It is hoped in the next number of the TRANSACTIONS to publish an obituary notice, together with a photograph, of Sir PATRICK MANSON, the first President of the Society, who has been well named the "Father of Tropical Medicine."

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Proceedings of a meeting of the Society held on Thursday, 16th February, 1922, at 8.15 p.m., at 11, Chandos Street, Cavendish Square, W. 1, preceded, at 7.45, by a Demonstration of Relapsing Fever Spirochætes in Lice,

Sir JAMES CANTLIE, K.B.E., F.R.C.S. *(President)*, in the Chair.

THE EPIDEMIOLOGY OF RELAPSING FEVER IN INDIA.*

BY

MAJOR F. W. CRAGG, I.M.S., *Central Research Institute, Kasauli.*

Relapsing fever and typhus have long been known to be associated with conditions of overcrowding, poverty and lack of personal cleanliness. We now know that these conditions are favourable to the spread of these diseases, because they are most favourable to Pediculus. The increasing rarity of both diseases in Europe in pre-war times is to be attributed to the gradual rise in the standard of living, and the corresponding rise in the standard of personal cleanliness, among the population. When, however, we come to consider the epidemiology of these diseases in India, we have to do with an entirely different set of conditions, and the problem is by no means so simple.

With the exception of the small fraction of the population which lives in the larger towns, the people are a simple peasantry, living by agriculture and the occupations directly connected with it. They are a conservative people, and though, with the security against aggression and against famine which they now enjoy, they are more prosperous, their habits and mode of life have probably changed little since the days of the Great Moghul. A comparison of Indian conditions with those of modern Europe is quite fallacious; overcrowding, judged by our arbitrary standard

*This paper when read before the Society was illustrated by Lantern Slides of Charts, which added greatly to the interest of the evening.

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of so many cubic feet per person, is practically universal, but this is not from want of space or from the expense of building, but from custom; the people may be poor in possessions, but their wants are few and simple. The essential fact, from our present point of view, is that the standard of living and of personal cleanliness has not reached that point at which man ceases to harbour Pediculus, his natural parasite. It is well to keep in mind that until a certain standard of cleanliness, not natural among primitive peoples, is reached, the presence of this insect is to be expected. That by far the greater part of the Indian peasantry normally harbour lice will not, I think, be disputed by those who have experience of the country. I convinced myself last year, by actual observations, that this is true in the United Provinces. The fact of the presence of lice, therefore, loses much of its significance in the study of relapsing fever in India.

When I first began the study of relapsing fever, a year ago, it appeared to me that there were considerable difficulties in the way of an explanation of the epidemiology of the disease on the basis of transmission by the louse. In the first place, the seasonal incidence is the opposite of what one would expect from published work on the bionomics of Pediculus, and from the accounts of the disease elsewhere. It is stated, and with good reason, that louse infestation is at its height in the winter, and, though authorities are not very emphatic on the point, relapsing fever, like typhus, is generally said to be a winter disease. In the plains of northern India, where the difference between the winter and summer climates is extreme, epidemics of this disease reach their height in the hottest months of the year, and are cut short before the onset of the rains. Secondly, the disease is not so generally distributed as one would expect it to be. Bengal and Assam, and the southern part of the Peninsula, as far as the available records show, do not suffer from the disease. It is especially curious that Calcutta and Madras should escape while Bombay does not. Further, whereas in other parts of the world typhus and relapsing fever are associated, as one would expect them to be, in India major epidemics of the latter occur without, so far as we know, any typhus at all, although typhus is almost certainly endemic in the Himalayas, and could in any case be very easily introduced into India through the passes leading from Central Asia. There are other considerations which tended to lead one's thoughts in another direction. It has

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been pointed out, in official communications which I was privileged to see, that the time of maximum prevalence of the disease coincides with the maturation of the spring crop. There is also a general impression that the disease is rural rather than urban. It happens also that the parts which have suffered most are the wheat-growing tracts. The disease is endemic in the Punjaub, which grows wheat, and apparently not in Bengal and the rice-growing tracts of Madras. In the United Provinces the western and central districts have suffered severely from the disease, and these are similar to the adjacent districts of the Punjaub, and grow wheat; the southern and eastern districts, which do not produce wheat, have never been so severely infected. Having in mind that relapsing fever is transmitted by two arthropods so dissimilar as the tick and the louse, a third vector, somehow connected with the wheat crop, appeared a possibility. It did not appear to me to be justifiable to accept the louse as the vector of the Indian disease without experimental proof.

The most important feature in the epidemiology of the disease in India, and the one most difficult to explain on the basis of louse transmission, is the occurrence, at long intervals, of very severe and widespread epidemics. Relapsing fever is undoubtedly endemic in northern India, but little is heard of it in normal times. In the Sanitary Reports of the United Provinces there are many consecutive years in which the disease is not mentioned at all. The major epidemics, which, fortunately, only appear once in a generation or so, rival the worst outbreaks of plague or cholera.

We learn from the old records that there was a severe epidemic in 1836 and 1837, another in 1862 and the following years. The next epidemic was that of which VANDYKE CARTER has left us such an admirable account. It was associated with the great famine of 1877 and 1878, and many of the cases seen by CARTER in Bombay were immigrants into the city from the famine-stricken areas in the north. Since then there have been many minor outbreaks recorded, particularly in the Punjaub, but none of these is comparable with that of 1877 and 1878. In the United Provinces the disease was recognised as epidemic in the western districts in 1911 and the following years, but there was no very marked effect on the total mortality as compared with the mean, a circumstance which suggests that the increased prominence of the

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disease was due rather to a better recognition of minor outbreaks than to an increased prevalence.

THE 1917 TO 1920 EPIDEMIC.

In 1917, after an interval of forty years, another great epidemic began. It reached its height in 1918, but was still active in 1920. It certainly affected the Punjaub, the United Provinces, and the Central Provinces, and may have extended elsewhere. My own observations have been confined to the United Provinces, and the account of the epidemic which I now offer refers only to that area. An account of the disease in the Punjaub has been prepared by GILL, and will, I understand, be published shortly.* Unfortunately, he and I have worked by different methods, and our results are not easily comparable.

A preliminary word is necessary about the Indian vital statistics. There is, in the ordinary routine, no registration of the cause of death by medical men. The diagnosis, such as it is, is made by the friends of the deceased, and reported by the village watchman, who is required to distinguish by name only three diseases: plague, cholera and small-pox. Other causes of death are reported under the general headings—"respiratory disease," "diarrhœa and dysentery," "fever," and "all other causes." The reports are collected by districts, and eventually appear, duly tabulated, with the Annual Reports of the Sanitary Commissioners of Provinces. It might be thought that statistical material collected in this manner would have little value, but this is not the case. A great deal of valuable information can be obtained from it, if it is studied with a knowledge of local conditions and of the manner in which the figures are collected. The total number of deaths, the seasonal variations in mortality, the age at time of death, and the sex are reported with sufficient accuracy. The census reports provide a control for the general figures, and special investigations made from time to time control the diagnosis of the named diseases. On the whole, the village officials appear to carry out their task remarkably well. The statistics are satisfactory in practice because one is working on broad lines, dealing with only a small number of major diseases, any one of which may, for a few months, double and treble the death-rate. It is very necessary to appreciate how large a proportion of

*The writer had the privilege of seeing Colonel GILL's paper in manuscript.

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the total number of deaths is due to epidemic disease. The mean annual death-rate in the United Provinces for the 20-year period 1898-1917 was 36·27; the lowest rate was 27·38, the highest 52·73, the high rates being due entirely to prevailing epidemics. The most important diseases are, of course, plague, cholera and malaria; small-pox is now relatively unimportant, vaccination being an established and not unpopular institution. The seasonal prevalence of these diseases is an important point, particularly in the case of plague, as it happens that the plague season is near that of relapsing fever. Plague is at its lowest in July and August; the death-rate from this disease rises during the cold weather, and reaches its highest point in March or April, according to the climate of the locality. Relapsing fever epidemics rise and fall about a month later than epidemics of plague.

The mean annual mortality for the 20-year period 1898-1917, when charted by months, shows two rises, the first including the months from March to June, the second the autumnal months. The autumnal rise is, of course, due to malaria. The first rise is partly accounted for by plague, but this cannot account for the whole of it, for we know that there is little plague in May in the United Provinces; and, further, the rise is shown in a modified form in the 20-year period before the present outbreak of plague. The great variability of the death-rate from year to year, and the part which seasonal epidemics play in the production of the totals, is best brought out by calculating the standard deviation of the figures from which the 20-year mean is obtained. The autumnal months show the highest deviation, indicating the great variability of malaria from year to year. The figures for March and April are high on account of the varying intensity of plague. The figures for May and June are lower, which suggests that the prevalence of relapsing fever has been much the same in each of the twenty years.

I may add that the lower death-rates shown in the earlier period are to be attributed to less efficient registration, and that the deaths recorded each month from "fever" run generally parallel to the total deaths from all causes, except in the presence of epidemic plague or cholera.

We now pass on to consider the recent epidemic as it is reflected in the vital statistics. In the first quarter of 1917 the death-rate was practically normal. In April, however, there was a very definite rise, which was almost maintained in May. There was not enough plague or

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cholera reported to account for this. The death-rate in the following months ran parallel with the mean, but was maintained above it till September. It is necessary to remark here that the autumnal rains failed in 1917, and that there was therefore little malaria. A sharp rise in December brought the death-rate to about 50 per cent. higher than in the first month of 1917. This rise was maintained in January, and from March the rate rose so sharply that in May, 1918, the death-rate was about 75 per cent. greater than the mean for that month. Thereafter the death-rate fell steadily until it reached the mean in August. We cannot follow the epidemic of relapsing fever through the rest of 1918, or in 1919, on account of the prevalence of influenza, which obscured all traces of other epidemics. The year 1920 was a most exceptional one, in that very little plague or cholera was reported. Relapsing fever was, however, still prevalent, and shows itself in a rise in May and June, rather later than usual.

The epidemic was not evenly distributed over the Provinces. The western districts, which are situated on the fertile plain between the Jumna and the Ganges, and are generally accounted the most prosperous in the Provinces, suffered the most. The death-rate in these districts at the height of the epidemic was more than two and a half times the mean for the same area. These districts suffered little in 1920.

Next to the seasonal prevalence, the most striking feature of the epidemic was the special incidence on young adults. I worked out the figures from the statistics of the Agra district, omitting the large towns, and taking the mean of the five years from 1912 to 1916 as a standard for comparison. The total deaths in each month are shown in vertical columns, divided into blocks representing the deaths at each age period. When the figures are thus arranged, it is seen that the deaths of infants and young children take little part in the production of the hot weather rise, this being due to an excess of deaths of adults. This is the case both in the mean and in the epidemic figures, but during the epidemic the feature was very greatly exaggerated. In the mean the deaths of infants and young children made up rather less than half the total deaths in May, whereas in 1918 the same age groups made up only a fifth. The point is brought out more clearly if we compare the deaths at each age group at successive stages of the epidemic, expressing the number of deaths for convenience as the number of times the mean. Taking the

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age period twenty to thirty years, in December and January the death-rate was nearly five times the mean, in February and March about seven and a half times, and in April and May over fifteen times the mean. During the same periods the death-rate of children under one year was increased to less than one and a half times the mean, less than two, and just over three times the mean. The deaths from relapsing fever of infants and young children were probably much fewer than these figures indicate; the death-rate in May was 196·68 per annum, and of those who died, or were seriously ill, many must have had young children, for the parent age was most affected. Many children must have died from inevitable neglect.

The sex incidence of the disease is a little difficult to work out, as there is normally a considerable excess of males in the population. It is clear, however, that males suffer more than females. According to my calculation there should have been 108 males living in this district to every 100 females. In May, 1918, there were 128 deaths of males to every 100 deaths of females at the age period twenty to forty years.

As was to be expected from the preliminary considerations already commented on regarding louse infestation, caste and occupation were found to have no very marked effect on the incidence of the disease. The lower castes are probably most affected in small epidemics, but the prosperous agricultural castes do not escape.

An important problem presents itself with regard to the origin of the epidemic. Did it reach the United Provinces from elsewhere, or did it arise from a focus, or from many foci, within the Provinces? There is little doubt on this point. We have direct evidence of the existence of relapsing fever in many of the districts previous to 1917, and particularly of its prevalence in those districts which suffered most in 1917 and 1918. A rise in the death-rate at the season at which epidemics of this disease normally reach their height is a constant feature of the statistics. There is, therefore, no reason to assume that the epidemic originated elsewhere. A detailed study of the figures relating to the Agra district confirmed the view that the epidemic originated, as does an epidemic of malaria, from innumerable local foci. I could find no indications of any special focus in the district, or of any invasions from the surrounding districts. The conclusion is unavoidable that some common cause produced the epidemic from innumerable foci.

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Although all the districts which were severely infected showed an increased death-rate in the last three quarters of 1917, the maximum intensity of the epidemic was reached earlier in some districts than in others. By the use of a method based on the seasonal prevalence and the special age incidence of the disease, I was able to estimate the relative severity of the disease in any district, irrespective of the total death-rate, which might, of course, be raised by an epidemic of another disease. I may add that I found this method most useful in the analysis of the statistics of past years, and by its aid was able to define an epidemic in 1896 which passed unrecorded at the time. The method fails in the presence of much cholera, and only one of the factors could be used in the years 1918 and 1919, on account of the prevalence of influenza. By the use of this "relapsing fever index" it was found that Shahjahanpur, to the east of the Ganges, was the most heavily infected district in 1917, the next in order being grouped round it, with an extension across the river to the Jumna-Ganges Doab. In 1918, Aligarh suffered the most, together with the surrounding districts in the southern part of the Doab. The prevalence of cholera in the hot weather in the southern districts obscures the situation. In 1919, the Doab districts were free, but there were indications of the presence of the disease in the eastern districts. In 1920 the disease was nowhere so severe, and the index was positive in only a few of the central and eastern districts.

The duration of severe epidemic conditions in any one district appears to be about two years. In April and May, 1917, the death-rate in Shahjahanpur was about three times the mean for that month, while in 1918 it was about twice the mean. In Aligarh the rise in 1917 was not very great, but in the following year the death-rate, at the height of the epidemic, was about five times that of the same month in a normal year.

Epidemics on so vast a scale call for more explanation than can be provided on the lines which have sufficed in the case of outbreaks in Europe. There was no change in the condition of the people at the time the epidemic began, no exceptional poverty, no special movement of the population. There was scarcity in 1918, on account of the failure of the monsoon of 1917, and scarcity conditions lead to an increased movement of the population, which may have been a contributory cause; but it is clear that the epidemic was well established before the

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scarcity, which in any case was only slight as compared with the famines of other days, was felt by the people. There must be some additional factor, up to now unrecognised, in the epidemiology of the disease.

EXPERIMENTAL WORK.

I have already indicated the considerations which, at the beginning of my work, led me to doubt the rôle of the louse in the Indian disease. My first task was to endeavour to repeat the experiments by which the mode of transmission of the disease was demonstrated by the French workers. I met with many difficulties. Monkeys proved hard to get, on account of caste prejudice, and though I had taken all possible steps to get news of local causes at Agra, where I had arranged to work, I failed to find a single local case from which to infect a monkey directly. I eventually found an outbreak at Rohtak, in the Punjaub, about eight hours' journey from Agra, and brought back as many lice as possible from cases and convalescents. It is very difficult to keep lice alive in the hot weather, and casualties were many.

The lice were kept alive, mainly in wristlets, for the period which seemed most likely to give a successful result, and were then emulsified and injected subcutaneously into monkeys. Altogether 109 lice, the survivors of a much larger number collected at Rohtak, were divided between five monkeys. None of these developed a definite illness. The temperature charts were not much use as a guide, for the animals were then freshly caught and very wild, and during the first few weeks the excitement produced by the necessary manipulation was enough to raise the temperature. Later on, however, infective blood became available, for I developed the disease myself, ten days after a long and tiring morning spent in infected villages. Through the kindness of my medical attendant, 2 c.c. of blood containing spirochætes was injected subcutaneously into a fresh monkey. The temperature chart showed two rises, only the second of which, I think, is to be attributed to the infection. When I saw the animal on the sixth day it appeared to be definitely ill. Under anæsthesia I obtained 10 c.c. of blood from its heart, and of this 5 c.c. was injected subcutaneously into a fresh monkey, and 1 c.c. into each of the five which had previously been treated with lice. The monkey which received the 5 c.c. duly developed the disease. Of the other five, two had a definite two-day fever, with spirochætes in the

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blood. Two had less definite rises, and unfortunately blood films were not taken systematically. The fifth monkey was a small one, a few months old. Though it had received the same dose of the same blood which had produced a definite infection in older and larger animals, the temperature only reached 104° F. on one evening, and no spirochætes were found in films made on that occasion. This seemed suspicious, and thick films which had been made previous to the injection of the blood were searched thoroughly. In a film made on the seventh day after treatment with possibly infected lice three spirochætes were found. This animal had received an emulsion of seventeen lice, removed six days previously from a patient seven days after the first relapse; in addition to the subcutaneous injection a large drop of the emulsion was placed in the conjunctiva. The temperature after this treatment was irregular, and on the fifth and sixth days was maintained at 104° F. It seems probable that there had been a mild infection from the treatment with lice, and that this had conferred on the animal sufficient immunity to enable it to resist the subsequent dose of infective blood.

The dissection of lice from cases of the disease, and after a single feed on an infected monkey, gave a more satisfactory confirmation of the work of the French investigators. The insects were dissected at intervals of from two to seventeen days after the last infecting meal. Of 67 lice from cases of the disease or from convalescents, spirochætes were found only in those of the seventh and eleventh days. Of 58 lice which had fed once on an infected monkey, spirochætes were found in those of the eighth, ninth and eleventh days.

The results are thus sufficient as provisional confirmation of the well-known experiments. I hope to have the opportunity of carrying out a more regular series of experiments at a future date.

THE BIONOMICS OF PEDICULUS UNDER HOT WEATHER CONDITIONS.

Accepting the louse as the vector of the Indian relapsing fever, we have to resolve the difficulties which I have already commented on. The solution of these difficulties is naturally to be looked for, in the first place, in the bionomics of the insect host, and I therefore carried out a large number of experiments, mainly directed to ascertain the reaction of the louse to the climatic conditions of the Indian plains in April and May. The

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normal mean maximum temperature in the United Provinces in January, the coolest month, is 73° F.; in February, 77·4° F.; in March, 89·8° F. The hot weather may be said to begin about the middle of April, the normal for this month being 100·8° F. May is the hottest month, with a normal of 104·9° F. Towards the end of June there is an increase in atmospheric humidity owing to the advance of the monsoon, and the mean for this month is 101·3° F. There is thus a difference of about thirty degrees between the maximum temperatures of the coolest and the hottest months. These are, of course, the official records, taken under special conditions. To obtain data representing more closely the conditions under which Pediculus lives, I made a series of observations in an outhouse arranged to represent, as nearly as could conveniently be done, the conditions of an Indian dwelling. As might be expected, the humidity records were much higher than the official ones, the maximum temperatures lower and the minimum higher.

The many and varied experiments led to one definite result. In the cold weather, when the atmospheric temperature is definitely lower than the body temperature, the bionomics of Pediculus are the same in India as in Europe. The warmth of the body is necessary to it. Development is retarded if it is separated from its host at night. But as the temperature rises rapidly at the end of March and in April, the conditions are changed. The temperature and humidity of an ordinary Indian house become practically the optimum for this insect. It is to be noted that atmospheric conditions are far more important to lice living on the lightly clad Indian than they are when the host is a European, with many thicknesses of cloth between the skin and the external air. When, therefore, the temperature of the air approximates to that of the body, there is no slowing down of the metabolism of the insect if temporarily separated from its host. It becomes an exceedingly active insect. In one experiment which I carried out to test the reaction of lice to sunlight, the insect travelled altogether 32 feet 6 inches on a sheet of Bristol board, on one run achieving 17 inches in a minute, a speed far greater than previous records. Eggs are laid more freely, the incubation period is shortened, and the period of growth is reduced. As the season advances, however, the optimum is passed, and the more rapid metabolism is associated with an increased mortality. Towards the end of April and in early May the lice begin to die off, and I found in the United

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Provinces, as was found in Mesopotamia, that they become very scarce when the hot weather has set in. Experiments carried out on the laboratory bench at that season gave much the same result as those carried out in incubators at home.

There is thus a cycle each normal year. During the cold weather the life history of the louse is much the same as in Europe, except that it is more susceptible to atmospheric conditions, being generally only separated from them by one or two thicknesses of cotton cloth; in the early part of the hot weather there is a period which is specially favourable to the insect; the extreme dry heat of May kills off all but a few. The application of this to the seasonal prevalence of relapsing fever needs little comment. The rise and fall of the disease is apparently determined by much the same climatic conditions as control plague, but, owing to the longer incubation period, the greater duration of the disease before death, and the period which must elapse before the louse becomes infective after the infecting meal, the death-rate from relapsing fever rises and falls later than that from plague by about a month.

THE EXCITING CAUSE OF THE RECENT EPIDEMIC.

The seasonal prevalence of relapsing fever in India is thus explained by the reaction of the insect host of the spirochæte to a varying climate. We have next to account for the origin of the great epidemics.

The explanation, when one has arrived at it, is very simple. The year 1917 was an altogether abnormal one. In the first quarter of the year there was nothing very unusual in the climatic conditons, but in April the mean maximum temperature was only 95° F., against a normal of 100·8° F. In May it was 95·5° F., which is nearly ten degrees below the normal. In June there was a deficiency of 3·3° F. The atmospheric humidity was in each of these months correspondingly high. The bearing of this on the incidence of relapsing fever is clear. The period during which optimum conditions for Pediculus prevailed was greatly extended, and the natural delousing which takes place in a normal May was greatly reduced. The endemic disease, not being cut short as it usually is, got a firm hold, and the number of cases continued to increase throughout the succeeding cold weather; when the period of optimum conditions arrived in the early months of 1918 it assumed the proportions of a formidable epidemic. The weather in May

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and June of this year was normal, but one natural delousing did not suffice to stamp out so intense an epidemic, and it was not till 1921 that normal conditions were restored.

The hot weather conditions of 1917 are described in the official *Monthly Weather Review*, as "unique," and the writer goes on to say that the only years comparable with it are 1877, 1878 and 1885. No comparison could be more apt for our purpose, for the years 1877 and 1878 were those of the last great epidemic of relapsing fever. Enquiring further into the matter, we find that the Meteorological Reporter, commenting on the conditions of 1877, points out that the exceptionally low temperatures during the first half of that year were confined to northern India. He attributed the cold wave to the unprecedented snowfall in the Himalayas during the preceding winter. The temperatures in the south, including Madras, were higher than usual. Now, VANDYKE CARTER makes a point of the fact that, though Madras suffered equally with the rest of the country from the famine, relapsing fever was not prevalent there.

As regards 1885, I had only to do with statistics of the United Provinces, and there is no definite evidence in these of an epidemic that year; I have not seen the weather report, and am unable to say how the cold wave year was distributed. I have little doubt, however, that further study will show that the same cause produced the same effect wherever it acted. I have mentioned an epidemic in the United Provinces in 1896, an event which passed unrecorded at the time. I was not able to obtain the weather report—these old reports are difficult to get—but from the remarks of the Sanitary Commissioner it is clear that similar conditions of climate were responsible for the epidemic.

The low temperatures of 1877 were attributed to the excessive snowfall in the winter; the similar conditions in 1917 to excessive rainfall in the spring. In each case the rains failed in the succeeding autumn. There was a similar failure in 1896. The relation between famine and relapsing fever is not that of cause and effect, but it seems probable that they are to be traced to a common cause.

The practical importance of this conclusion is very great. Of all the diseases with which the sanitary officer in India has to do, none is more elusive than relapsing fever. The onset of the epidemic is slow, insidious, unobserved. The people, so accustomed to fever, are apathetic, uncomplaining, and unlikely to report a malady which has nothing dramatic

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about it to attract their attention. The seasonal prevalence is near that of plague, and the most severely infected areas are often also plague stricken. But with a knowledge of the effect of climate on the prevalence of the disease we are forewarned. An exceptionally cool summer should lead to a critical examination of the monthly vital statistics for any excess deaths of young adults, and a local investigation and the examination of a few blood-films will settle the matter long before the epidemic reaches serious proportions.

I may add a word regarding the causes underlying the special incidence of the disease among young adults. I think this is connected with the mode of infection. NICOLLE says that all infested persons scratch themselves, and explains infection as occurring through the act of scratching, the integument of the louse being ruptured and the infective body juices inoculated by the same act. I doubt if this is the ordinary event. The integument of the louse, as a little experiment shows, is tough, and the amount and kind of force applied in casual scratching is not very likely to rupture it. Moreover, in India at least, infested persons do not scratch themselves much or vigorously. Having harboured these parasites all their lives, they are not much annoyed by them. Infants and young children are very unlikely to infect themselves in the way NICOLLE suggests, and this may explain their relative immunity. But it appears to me more likely that infection is normally brought about by the common practice of crushing the louse between the finger and thumb-nail. The body juices are thus brought into contact with the soft tissues under the nail, through which the spirochæte may be able to penetrate. In any event the nail is infected. The very young cannot delouse themselves in this way, and the very old are too indifferent.

The sex incidence may be explained, as has been suggested, I think, by BISSETT, by the greater liability of the males, who are mainly engaged in hard manual labour, to abrasions on the hand.

CONCLUDING REMARKS.

There are many questions still awaiting investigation. One of the most important of these is the prevalence of the disease in normal years. It appears probable that relapsing fever is responsible for a considerable part of the rise in the death-rate in April and May, which is a normal

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feature of the vital statistics of northern India. The experience of those who have worked in other similar climates would be most interesting. One asks, as a preliminary question, Is extreme heat *per se* unfavourable to the life of the inhabitants of such climates?

The variation in the virulence of the infection is a subject of interest and importance. The variability, as shown by Indian experience, is very great. The virulence seems to be highest when the cases are most numerous, and it is possible that it is exalted by rapid passage. If the virulence becomes correspondingly reduced when the disease is quiescent, relapsing fever may be one of the perplexing group of "short fevers." CASEAUX met with cases so mild that the patient was able to continue his ordinary occupation.

The geographical distribution calls for investigation. The absence of the disease, or, at any rate, its rarity, in Bengal and Madras, may be accounted for on general grounds, such as the greater cleanliness of the people, the habit of anointing the body with oil, the very scanty clothing worn by many of the agricultural castes, etc., but such explanations do not appear wholly satisfactory. A study of the variations in the virulence of the infection, and of the nature of the immunity conferred by an attack of the disease, suggests itself as a promising line of enquiry. We know that the immunity after an attack does not last long. The seasonal prevalence of the disease has been traced to the seasonal changes in the climate of northern India. The cutting short of the epidemics in May must be followed by a progressive loss of immunity, and from the information we possess we must assume that even those who had a severe attack are again susceptible the following spring. Now in the parts with a more equable climate, which I may add are also the rice growing tracts as a rule, there is no such prolonged period during which climatic conditions render epidemics impossible. If the disease exists there, one would expect it to be in a mild form, for the people have no chance of losing their immunity to the infection. On such a hypothesis one would expect serious epidemics of relapsing fever to occur not in hot or cool climates, but in alternating climates, where the hot season is sufficiently pronounced almost to stamp out the disease, and thus to cause the population to lose their immunity.

The apparent absence of typhus is perplexing. The suggestion that

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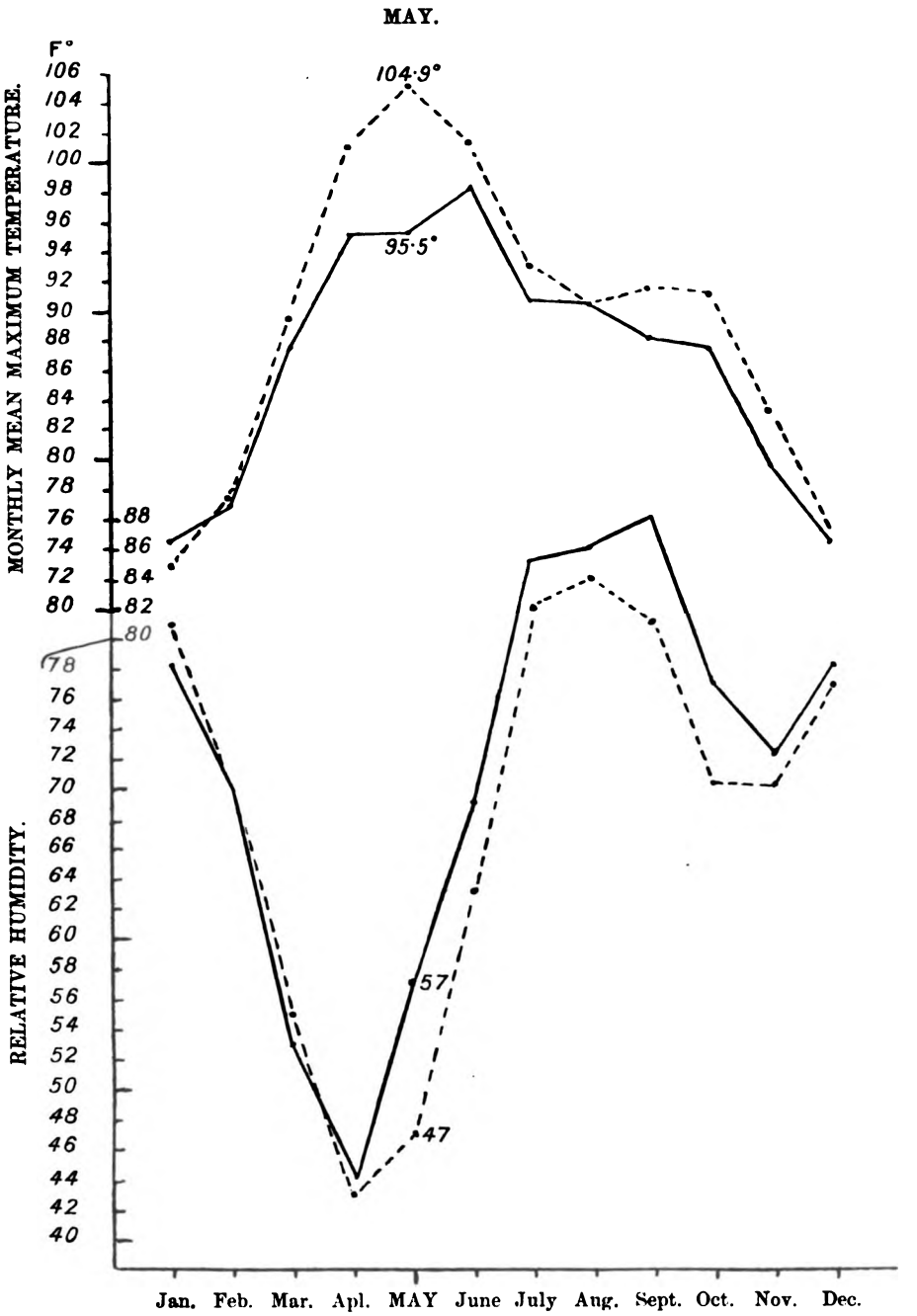

Temperature and humidity in the United Provinces in 1917 as compared with the normal.

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the typhus organism can only develop under certain climatic conditions arises only to be dismissed, for until the atmospheric temperature is near that of the body it can hardly affect Pediculus. The possibility that it has been present, but not recognised, cannot altogether be put aside, for many medical officers must be under the disadvantage, as I am myself, of having had no experience of this disease. We know from the records left by the past generation, who had better opportunities of seeing the disease in their student days, that it was endemic in the Kumaon hills, and there is no reason to suppose it has died out. At present we are confronted with a curious phenomenon, that two diseases with the same insect vector have an entirely different seasonal prevalence and distribution within the borders of the same country.

In conclusion, I should add that a full report of the results of my enquiry into relapsing fever in the United Provinces of Agra and Oudh will, I hope, appear in due course in the *Indian Journal of Medical Research*. In this brief communication I have avoided dwelling on the statistical and experimental detail.

Discussion.

The President: We have just listened to a deeply interesting lecture, one made all the more interesting by the fact that Major Cragg did not read from his manuscript, but gave us of his own experience straight from his mind. I am sure you will feel thankful to him for the lucid way in which he has stated his case; it has been a great treat to us all. There are probably some here who have never seen relapsing fever, but I know there are some present who have, and we shall be glad if they will give us the benefit of their experience and impressions in connection with the disease.

Major H. C. Brown: I have been very interested in Major Cragg's lecture to-night, especially as I was sent down, in 1913, to investigate an epidemic of relapsing fever in the Meerut district also. What struck me most there was that it appeared to be particularly rife in the rural districts rather than in the towns. If one asked the civil surgeons what was their experience, they said: "You may get a few cases in the towns, but you

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will not meet with it there in epidemic form, and there is no real severity about it." The villagers said that they did not know what the cause was, but they were certain it was something to do with the crops. Major CRAGG had mentioned that the disease occurred in the wheat-growing and not in the rice-growing districts. When people went into the fields to cut the crops, he noticed that when they took off their spare clothing before starting the work, they did not put it singly on the ground but in piles, which became even three feet high; and when the sun shed its rays on the clothing, the lice apparently made their way from the top to the cooler clothing beneath, thus bringing about a pretty thorough circulation and infection. This may explain to some extent the bigger proportion of cases in rural districts in comparison with the towns.

There was another possibility, which I suggested at the date when I was there, namely, that another vector might be one of the pentatomid bugs. During the mustard crop in May I counted, on a single twig of that plant, forty pentatomids. And I have seen the bug on threshing-floors, with many convalescents about, and I have noticed patients drawing up their legs and sweeping the bugs off because they were biting them. MARKHAM CARTER has proved that another pentatomid is capable of biting man, and I think some more work on this is required.

With regard to the sex incidence, I compiled a fair number of statistics there, and examined many more; and I found that not only was there a rise in relapsing fever in May, but also a December rise. I also found that in the May and December rises the males considerably outnumbered the females in the mortality from fevers.

Colonel H. J. BARRATT: I would like to make a few remarks with regard to the excellent paper which has just been read. I was in India at the time of the last relapsing fever epidemic which Major CRAGG has described, in 1917 and 1918, when I happened to be serving at Meerut. We had numerous cases at different stations: Saharunpur, Agra, Delhi, and other places, but it was in Meerut and Delhi that we had the largest incidence of cases, and, though I am speaking especially of military people, it was prevalent among the general population as well. As Major CRAGG says, it was in April and May that we had the very high death-rate. We had to supply a large number of coolies for the Labour Corps in connection with the War, and this necessitated a great deal

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of movement among that class of the population, and we found they suffered very much from the disease.

We had enormous camps at various places, and thousands were passing through them every month. Some of these men seemed to have brought the sickness with them, and once such cases arrived in the camp, and proceeded to mix with others, it was easy to see how the disease was disseminated. We had to take the greatest care, therefore, in forming segregation camps for these Labour Corps men, where they could be examined and cleansed, and clothing and bedding disinfected, before mixing with the general camp population. But among the labouring classes there was one thing which struck us forcibly—it happened at Delhi and also at Meerut. New huts were being erected, built of brick, and with a thatched roof. The work was done by civil labour and coolies employed who had suffered from the disease. It was unavoidable—there was no alternative. Indian troops who had not had relapsing fever and who occupied these huts got the disease, and the question arose as to how they became infected. It was found that the coolies had brought the lice with them in their clothing and bedding, and had left some of them in the huts, so that when healthy men went to live in these huts they became infected. As soon as an infected case occurred, we isolated him, segregated the contacts, and disinfected his clothing and bedding as well as the place as far as possible. Owing to these sanitary preventive measures the disease eventually disappeared. There is no doubt that it is by the intermingling of large numbers of people that the widespread dissemination of the disease takes place, through the agency of lice harboured by convalescent or infected cases.

Colonel CLAYTON LANE: Major CRAGG has referred to Darjeeling, and has alluded to the relationship between typhus and relapsing fever. DUKES described an epidemic in Darjeeling which was brought in from Sikkim. In this epidemic the sufferers had high fever, an enlarged liver and jaundice, and there were a number of deaths. It was only a small epidemic. There were no relapses—cases either got well or they died. The blood of these people contained spironema, and the slides from those cases were quite as striking as those exhibited here to-night. That was an anomalous condition, and one recognised that it was a difficult matter to say exactly what the disease was. MACKEY said he could find no difference

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between this spironema and that which was found in Bombay responsible for the relapsing fever there. I think it is due to the memory of an able medical officer to bring before the Society these observations.

Dr. ALDO CASTELLANI: I am interested in the observation made by Major CRAGG that epidemics of typhus and relapsing fever, though the two diseases are carried by the same insect, may not occur together. My experience in the Balkans and in Serbia, in 1915, was as follows:—We had typhus and relapsing fever there—a very large number of cases—until the beginning of July. In July typhus stopped quite suddenly, and from the end of July until October or November we had no other cases. But relapsing fever continued through the summer, and we had a fairly good number of cases of it.

I would like to ask Major CRAGG whether he has made any investigation on how long the period of infectivity lasts? I have seen a very large number of cases of relapsing fever in the Balkans, also at Corfu and in Poland, and I have the impression that the period of infectivity is much longer than is generally admitted. I think that the blood of the relapsing fever patient remains infective for several weeks, possibly months, after the last attack of the fever. In Macedonia and Corfu I investigated ten cases carefully, and found in four or five of them that, after the last typical rise of temperature, the temperature chart was not quite normal for four or five weeks; every fourth or fifth, or every seventh or eighth day, there was a slight rise, perhaps no more than to 99°F., and the patient was not feeling well. We found spirochætes in only one of the cases, but my impression is that the blood remains infective for several weeks, possibly months, after the last definite attack of fever.

Lieut.-Col. J. C. KENNEDY: I can bear testimony to Major CRAGG's remark as to the prevalence of relapsing fever in the Kamaon district. I was stationed at Nainital, and we had the greatest difficulty in preventing relapsing fever being carried down to the plains by the coolies, of which Colonel BARRATT has spoken. We had large camps there, in which we collected the coolies who had been recruited for military duty. That was one of our greatest difficulties, to eliminate the carrying of relapsing fever from those parts down to the plains. From the military point of view, we had the idea that the relapsing fever which

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was prevalent amongst the Indian troops originally came from the Hill districts.

Major F. W. CRAGG: In reply to Major BROWN, who mentioned the difference in the incidence of the disease in rural as against urban areas, I tried to ascertain details about that, but I was unable to get anything satisfactory from the statistics. I had the impression that relapsing fever was a rural rather than an urban disease; but it was a thing one could not be certain about, for facts concerning it are not easily ascertained.

As to the rise in incidence in the cold weather, there is a very definite rise in the total mortality in December and January in the United Provinces. I could not get evidence that it was due to relapsing fever; it is generally attributed to pneumonia. I do not know that the suggestion of pneumonia would explain the sex incidence which Major BROWN mentioned. I would like to take this opportunity of expressing my thanks to Major BROWN for the many valuable hints I got from him before I started this investigation. He was there in 1913; my visit was later.

Dr. CASTELLANI mentioned that relapsing fever will go on longer than typhus, and I think an explanation of that may be found. If we were to delouse the entire population on one day, all the people who were going to die of typhus would die of it in ten to fourteen days. In the case of relapsing fever they might recover from the first attack, from the second, the third, even a fourth attack, and die from the next attack; and there is no doubt, as I found in my own case, that convalescence from relapsing fever is a very slow and difficult business, and any intercurrent disease coming along at that time would be a serious matter for the patient.

With reference to the period of infectivity, some of my experiments showed that lice can be infected from patients who have long recovered from their relapsing fever; but more observations are required on this point.

The PRESIDENT: I am sure you will join with me in thanking Major CRAGG for his very learned paper.

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COMMUNICATIONS.*

A NATIVE METHOD OF VACCINATION AGAINST SMALLPOX.

BY W. E. HAWORTH, M.B., C.M., *M.O.H., Tanganyika Territory.*

"On the 24th June, 1921, Thomas Saidi, a Bondei native, from Kwasemgaya, near Magila, called at the office to see one of the sanitary inspectors. Louis Mynah, clerk at the Health Office, to whom Saidi was well known by repute, brought him to the Medical Officer of Health, to whom he willingly gave an account of himself and his operations. He grows cassava on a shamba of his own, and also travels about the district adding to his income by inoculating people as a protection against smallpox. His fee is three cents, young children being operated upon free of charge. His outfit consisted of a common pocket-knife, a small pot filled with blue-black paste, and two bottles of a brown fluid. The last-mentioned he prepares from the fruit of two trees, one called mvungunya or mnyegea, the other mtenga. His method of preparing the paste is as follows: He takes some grass on which a person sick with smallpox has been lying, carries it in a cloth to the forest, and there burns it on a spot from which other grass has been cleared. He then gathers up the ash and mixes it with the juice of a tree called ngeza. After this he washes his body and clothes. In operating he disinfects the knife with fire, and with it he scarifies a place on the outside of the arm or front of the forearm, applies some of the brown fluid, and then rubs in a little of the paste. He states that two days after the operation the patient becomes sick with fever, which continues for four days, and that some of the patients become affected with smallpox afterwards, but have only mild attacks. On one occasion he operated on three men, and three weeks thereafter a child of one of them developed smallpox, and eight days later the father was attacked. Both were mild cases. The children of the other men also had smallpox, but, being away in another part of

*With a view to making the TRANSACTIONS more interesting, it is proposed to insert SHORT COMMUNICATIONS from Fellows relative to any matters of medical, sanitary or parasitological interest. It is felt that many Fellows in various parts of the world must be in a position to contribute short papers on these subjects, and the Secretaries will be pleased to receive any such from them.

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the district, he did not know how long it was after he had inoculated the fathers. His father taught him the method, and he wrote down the directions for reference till thoroughly conversant with them by practice. When smallpox was prevalent, and many people required his services, he earned as much as Rs. 2 in a week. Last year he had operated upon many people, and on the morning of the interview had done so on a clerk in the District Political Office.

"He appeared to be quite sincere in his belief that he protected against smallpox, and to have no suspicion that his procedure was likely to cause the spread of the disease. His outfit was purchased for Rs. 4. He was then sent to the District Political Office, where he received a warning to desist from his practice. Possibly his method is a descendant of a form of inoculating the disease introduced from the East or from Europe."

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A CHEAP FORM OF ARTESIAN WATER SUPPLY FOR VILLAGES IN THE TROPICS.

BY

E. P. MINETT, M.D., D.P.H., D.T.M. & H.

Government Medical Officer of Health, British Guiana.

The problem of a water-supply in British Guiana is not difficult as regards quantity, with a rainfall of about 100 inches per annum in a country the coastlands of which are four feet below high-tide level; but the quality of this surface water—conserved in shallow, almost stagnant, trenches and canals—leaves much to be desired. The water picks up about 4 grs. per gallon of peaty substances, and is of a brown sherry colour; in addition, these water trenches, running for miles, are liable to very serious pollution. The better-class houses are provided with vats or tanks for conserving rain-water, collected from the roofs; but this source of drinking-water is also open to many serious objections, both from the point of view of pollution and as lending itself to mosquito breeding.

About 1912, several artesian wells were sunk in the colony, in order to tap a pure water supply at considerable depth. The majority of these wells draw water from a depth of 500 to 600 feet. At first, considerable difficulty was experienced, owing to their gradually decreasing flow caused by silting up with fine sand. The well here described was fitted with a special strainer, designed by E. G. Buck, at that time Colonial Civil Engineer in the colony. The Nabaclis well is fitted with 8in. casing, and draws water from a depth of about 600 feet. The outflow has been fairly constant for the last six years at 250,000 gallons per day.

The water as it issues from the well is, however, not very palatable, owing to its containing a very considerable amount of iron in the ferrous state and, in addition, the temperature is about 95° F.; the water was, therefore, not popular with a people accustomed to rain-water cooled by storage in vats, barrels, etc., to a temperature of about 75° F.

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In order to render this artesian well-water palatable, it is necessary to remove the iron by free aeration and filtration, then to reduce the temperature by storage. Both these problems would be easy if funds were available to provide a large pumping and filtration plant, but only a small grant is available annually for this work. Mechanical plant is out of the question, on the ground of expense in installation and maintenance. As the country is dead flat the use of any form of gravity plant is precluded. The problem before the Public Works Department and the Health Department was briefly this, "How can we aerate, filter, cool and distribute this water when the water only rises about four feet above ground level in the well casing?" The attached plan shows the plant adopted, after consultation and on the advice of Mr. H. BEEBY THOMPSON, the Consulting Engineer, who visited the colony in 1920. The installation of this water supply is due to the generosity of Mr. A. DUCKHAM, who placed the sum of £1,000 at the disposal of the Government Medical Officer of Health for the purpose. The work was carried out by the Public Works Department, and when completed placed under the control of the Health Department and the Local Authority Many unforeseen difficulties arose during construction, not the least of these being that, in order to avoid "pitting" of the iron pipes, the whole of the 6in. mains had to be lined with bitumen, obtained from Trinidad at considerable expense. No motive power is used, and the plant is self-acting, the only working head available being the four feet to which the well-water rises above ground level, and the mains being laid at a depth of three feet below ground level, giving a total available head of seven feet. The water is raised from the main by means of pumps placed at suitable points throughout the village. The people, drawing the water themselves, supply the only motive power used throughout the system.

The explanation of the letters on plan will give the details of the working, perhaps, better than a written description. A very large quantity of iron is deposited before the water reaches the sand filter. It has been running for three months at present without cleaning the sand, but the surface of the filter is becoming thickly coated with deposited iron. In order to remove this, an arrangement for reversing the current through the filter has been fitted, and found to work satisfactorily. I anticipate that it will be necessary to clean the filter about every three months.

The following analyses were carried out by Sir J. B. HARRISON, C.M.G., who also supplied the appended certificate of purity, etc. We are also

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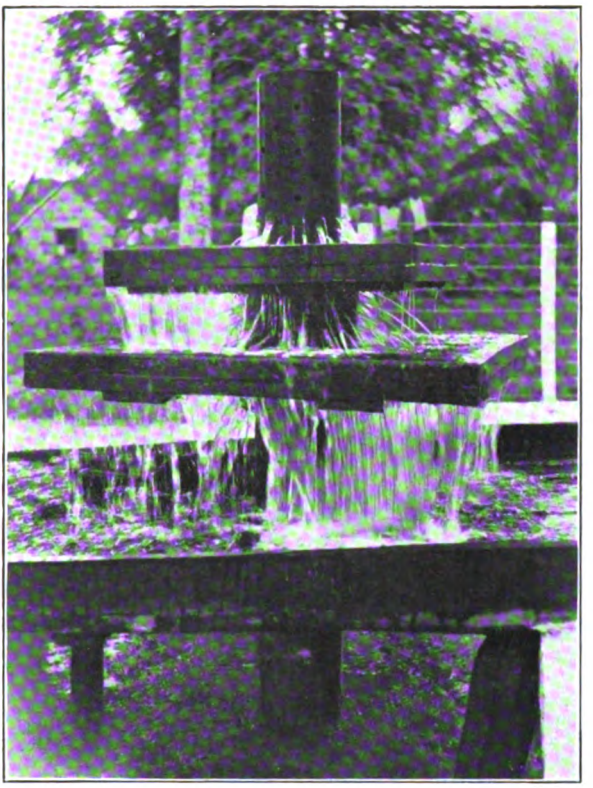
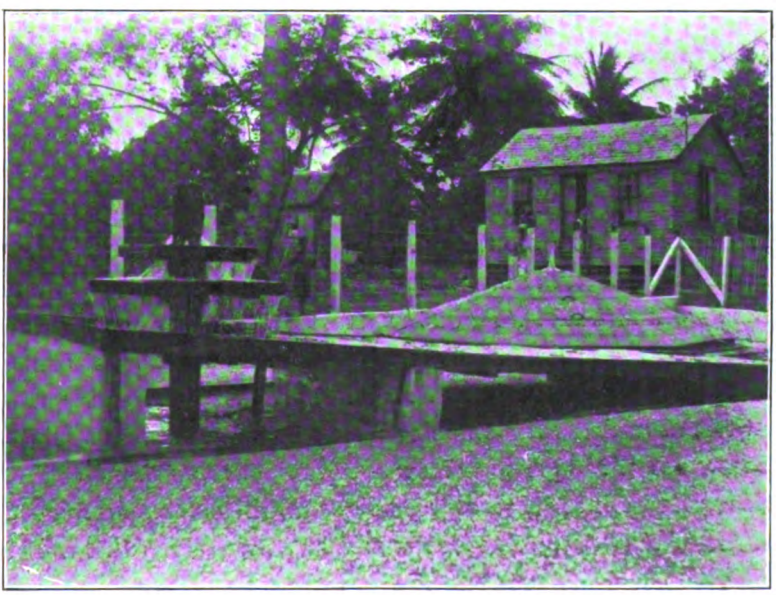


Showing perforated casing to well at water exit, with trays to spread out water and assist aeration.
Top of tray with baffle boards and large stones shown.



Spray, etc —Showing double roof of storage tank beyond.

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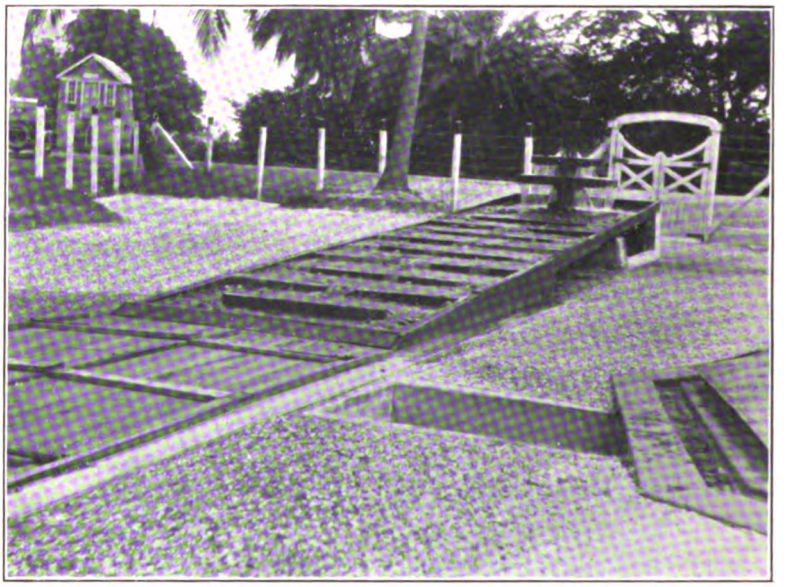

View of spray and inclined plane shoot with baffle boards and large stones to collect deposited iron. Filter shown with wood covering closed.

Type of pump and concrete platform used to distribute water at roadside.

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deeply indebted to him for many hints and suggestions for improvements during the construction :—

Samples taken by the Government Medical Officer of Health and the Director of Science and Agriculture on 9th December, 1921.

ELEMENTS AND IONS.	PARTS PER 100,000 OF WATER. *Water flowing from casing.*	*Water aerated and filtered as delivered from main.*
Iron	·56	Nil.
Calcium	·55	1·09
Magnesium	·56	·78
Sodium with traces of potassium ...	2·67	1·51
Chlorine	1·84	1·84
Sulphate ion	·06	trace
Nitrous ion	·007	·003
Silicate ion	·95	1·27
Carbonate ion	3·96	2·61
	11·157	9·103

SANITARY EXAMINATION.	*Water as falling from casing.*	*Water as delivered from main.*
Dissolved oxygen, per cent. of possible	58·2	87·4
Relative proportion of free carbonic acid in solution	100	33
	Parts per 100,000 of water.	
Oxygen absorbed from acid potassium permanganate at 82° F.		
In 3 minutes	·08	Nil.
In 4 hours	·15	·25
Free Ammonia	less than ·001	less than ·001
Albuminoid ammonia	,, ·001	,, ·001
Nitrogen in nitrites	·002	·001

The water of the Nabaclis water-supply scheme, as delivered from the main, is brilliantly clear, colourless, tasteless and odourless ; it is well aerated and palatable ; while it exceeds in purity that of any other water-supply known to me in the West Indian Islands or in British Guiana. It is quite free from iron, while the origin of the water from aquifers, 770 to 850 feet below the surface of the ground, ensures its freedom from all organic pollution.

24th December, 1921. *(Signed)* J. B. HARRISON.

It is now proposed to develop this system, and gradually provide the city of Georgetown and the towns and villages of the colony with a pure water supply on the above lines. Two of the larger sugar estates in the colony have also developed their artesian wells on similar lines, with slight modifications to suit local conditions. One estate raises the water, after treatment, into two large iron containers, and distributes the water throughout the ranges occupied by the labourers at a very small cost.

Considering the very high incidence of bowel complaints in the colony, generally due to defective water-supplies, I am certain that the gradual adoption of this form of water-supply will, from a health point of view, amply repay the initial cost.

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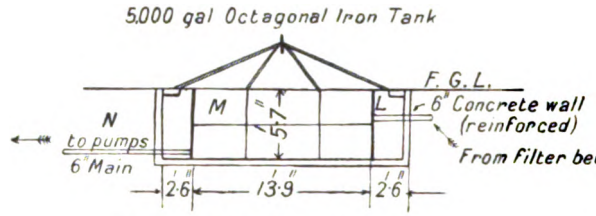
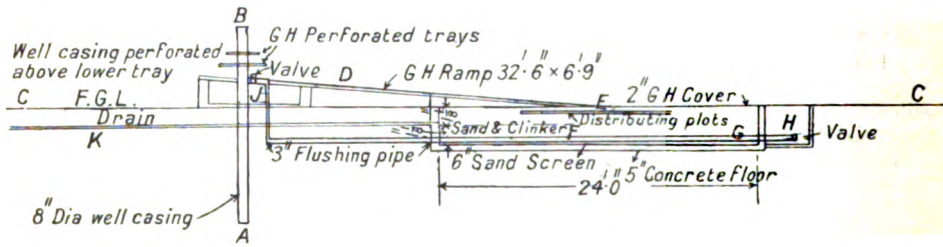


Sectional Elevation Y--Y.



Sectional Elevation X--X

EXPLANATION OF LETTERS TO PLAN.

A. Well casing up from depth of about 560 feet, pipe is 8 inches diameter.

B. Outlet of well perforated at sides to break up water and increase aeration to get rid of Fe. by converting it from ferr*ous* to ferr*ic* state.

C. Ground level: this shows what a small amount of *head* we had to work with—only about four or five feet. (Four or five feet is about all we ever get.)

D. Sort of shoot, with baffle boards arranged as shown on ground plan, *B*. Spaces between filled with stones to further break up and aerate water.

E. Flat concrete table, perforated ½-inch holes, and with edges raised one inch all round to further spread out and aerate water. *Also* to prevent water falling all in one spot of sand-filter surface, and thus destroying scum in surface.

F. Layer of stones, gravel and fine sand to remove iron rendered insoluble by aeration, also to serve purpose of an ordinary filter.

G. Perforated pipe fitted with external strainer, which collects filtered water from the bottom of sand and conveys it into the storage tank.

H. Stop-cock to shut off water from going into tank when it is desired to reverse the current to clean the filter. (Same principle as Jewell filter, only adapted to this.)

I. Stop-cock to open when water is desired to pass down pipe, *J*, and reverse current to clean filter. Waste water after flushing passes down drain, *K*.

L. Entry pipe from filter to storage tank, *M*

N. Exit from storage tank to 6in. mains running along the road three or four feet below surface.

O. Main in section showing reducing tee.

P. Pump 2in diameter up to pump.

Q. Pump above ground level for drawing water.

R. Concrete slab round pump for standing buckets, etc., on.

AERATION AND FILTER PLANT, ARTESIAN WELL, NABACLIS VILLAGE.

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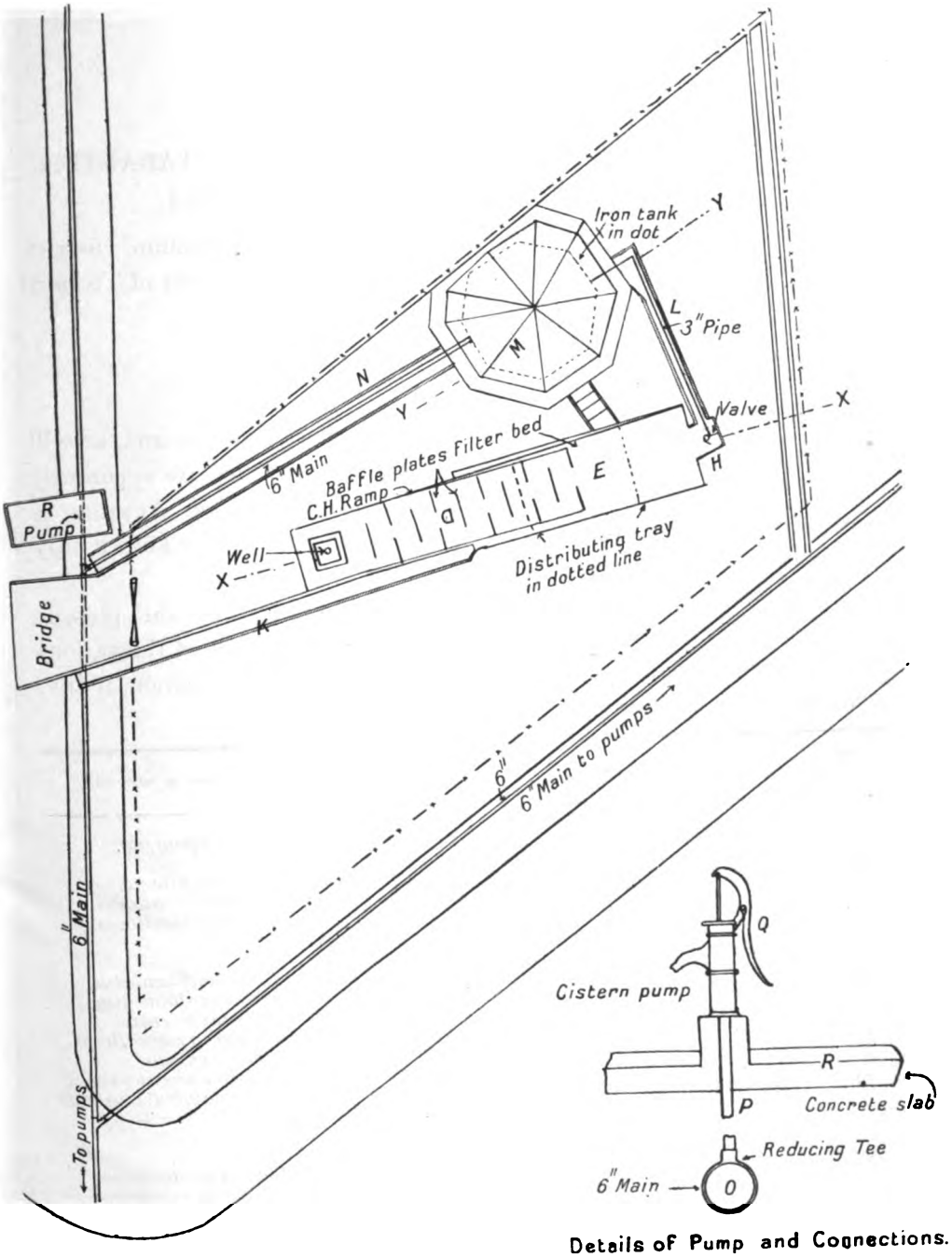

Details of Pump and Connections.

AERATION AND FILTER PLANT, ARTESIAN WELL, NABACLIS VILLAGE.

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NOTE ON A SMALL COLLECTION OF INDIAN TABANIDÆ AND OTHER BLOOD-SUCKING DIPTERA.

Formed by Captain H. E. CROSS, I.C.V.D., Sohawa, Jhelum District, Punjaub, and received for examination from the Royal Society of Tropical Medicine and Hygiene.

BY MAJOR E. E. AUSTEN, D.S.O.

The collection, though small, is of considerable interest, and, as will be seen below, contains examples of several species which are apparently new. The occurrence in India of *Lipoptena caprina*, AUSTEN, is especially worthy of note, since as yet this Hippoboscid is known only as a parasite of goats in Palestine.

The subjoined list, in addition to determinations by the present writer, also shows the names under which the examples of the various species were received, in order that differences in identification, if any, may readily be appreciated.

Serial No.	Family.	No. of Specimens	Name.	Name as received.
i.	Tabanidæ	1	*Hæmatopota* sp. incert., prob. nov.	*Hæmatopota*, sp.
ii.	,,	4	*Tabanus striatus*, FABR.	*Tabanus hilaris.*
iii.	,,	1	*Tabanus* sp., prob., nov.	*Tabanus bicallosus.*
iv.	,,	3	*Tabanus macer*, BIG. (Syn. *T. bicallosus*, RIC.; *T. trichinopolis*, RIC.)	*Tabanus bicallosus.*
v.	,,	4	*Tabanusditæniatus*, MACQ.	*Tabanus ditæniatus.*
vi.	,,	4	*Tabanus albimedius*, WALK.	*Tabanus albimedius.*
vii.	,,	2	*Tabanus consocius*, WALK.	*Tabanus orientis.*
viii.	,,	3	*Tabanus nemocallosus*, RIC.	*Tabanus nemocallosus.*
ix.	,,	3	*Tabanus virgo*, WIED.	*Tabanus virgo.*
x.	,,	2	*Tabanus*, sp. nov.	*Tabanus sexcintus* (sic).
xi.	Muscidæ	2	*Philæmatomyia crassirostris*, STEIN. (Syn. *Ph. insignis*, AUSTEN).	*Philæmatomyia insignis.*
xii.	,,	1	*Stomoxys calcitrans*, L.	*Stomoxys calcitrans.*
xiii.	,,	1	*Stomoxys* sp. incert., nov. ?	*Stomoxys calcitrans.*
xiv.	,,	1	*Lyperosia exigua*, DE MEIJ.	*Lyperosia exigua.*
xv.	Hippoboscidæ	2	*Hippobosca maculata*, LEACH.	*Hippobosca maculata.*
xvi.	,,	1	*Lipoptena caprina*, AUSTEN.	*Lipoptena* sp. near *cervi.*

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